Exhibit 10

to

Declaration of Declaration of Andrew S. Hansen

Ralph Simon v. Select Comfort Retail Corp.,
and Select Comfort Corporation
Case No.: 4:14-cv-1136 (JAR)

ERNEST P. CHIODO, M.D., J.D., M.P.H., M.S., M.B.A., C.I.H.
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EPC

Diplomate of the American Board of Internal Medicine
Diplomate of the American Board of Preventive Medicine
in Occupational Medicine
Diplomate of the American Board of Preventive Medicine
in Public Health and General Preventive Medicine
Diplomate of the American Board of Industrial Hygiene
as a Certified Industrial Hygienist

June 4, 2015

David S. Corwin, Esq.
Sher Corwin Winters
190 Carondelet Plaza
Suite 1100
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Tel. (314) 721-5200
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Re: Ralph Simon v. Select Comfort Retail Corp., et al.

Dear Mr. Corwin;

I have reviewed materials and conducted an independent medical evaluation on your client in order to determine if he suffered any injury or illness due to mold exposure in this matter.

QUALIFICATIONS TO OPINE

My qualifications include a Medical Degree (M.D.) from Wayne State University School of Medicine, a Master of Public Health (M.P.H.) from Harvard University School of Public Health, a Master of Science in Biomedical Engineering from Wayne State University, a Master of Science in Threat Response Management (biological, chemical, and radiological defense) from the University of Chicago, and a Master of Science in Occupational and Environmental Health Sciences with Specialization in Industrial Toxicology from Wayne State University. I am board certified in the medical specialties of Internal Medicine, Occupational Medicine, and Public Health and General Preventive Medicine. I am also certified in the engineering and public health discipline of industrial hygiene by the American Board of Industrial Hygiene as a Certified Industrial Hygienist (C.I.H.) in the comprehensive practice of industrial hygiene. Industrial hygiene is the discipline involved in quantifying and controlling occupational and environmental hazards including air toxins including mold. I am a past president of the Michigan

Industrial Hygiene Society which was the first industrial hygiene organization in the United States. I served for many years as an assistant clinical professor of internal medicine, family medicine, and public health at Wayne State University School of Medicine as well as an adjunct assistant professor industrial hygiene and toxicology in the Department of Occupational and Environmental Health Sciences at Wayne State University. I have served as the Medical Director and Manager of Medical and Public Health Services of the City of Detroit and was the chief physician responsible for measures designed to protect the public health of over one million persons living or working in the City of Detroit during the time of my service. I am licensed to practice medicine in Michigan, Illinois, Florida, and New York. Since I have had a long standing clinical interest in building related disease including mold exposure, I have maintained a residential builder license in the State of Michigan since 1990.

RECORDS REVIEWED

1. Medical records from SSM Medical Group and Dr. Thomas Spiro.
2. Medical records from Dr. Phillip Martin.
3. Medical records from Dr. Robert Craig.
4. Medical records from American Sleep Medicine.
5. Medical records from Wilmington Clinic. Medical records from Dr. Theodore Sullivan.
6. Medical records from Dr. Mark McCarthy.
7. Medical records from Dr. Wedner.
8. Mold report by Fungus A Mungus dated August 1, 2013.
9. Report by Microbe Inotech Laboratories, Inc. dated March 26, 2015.

HISTORY

Ralph C. Simon (D.O.B.: [redacted]1956) was well prior to buying a Select Comfort bed. About three or four years after buying the bed Mr. Simon began suffering recurrent sinusitis, ringing in his ears, a sensation of pressure in his bilateral ears, dry and burning eyes, and skin irritation. He also suffered from exertional shortness of breath. Also had general malaise. He sought medical attention for his conditions. It was the impression of Dr. Craig, Dr. Martin, and Dr. Sullivan that he was coming into contact with some material to which he was allergic. They questioned him about possible mold exposure. However, no source of mold was identified at that time. Due to his symptoms and clinical presentation, he was prescribed prednisone and antibiotics that included gentamycin. He developed loss of the sense of smell and permanent hearing loss in the left ear. While he had some hearing loss in the left ear prior to taking gentamycin, his hearing loss in the left ear became profound after taking gentamycin. He also developed

a rapid onset of bilateral cataracts at the age of about 53. Neither he nor his treating physicians knew of the mold contamination of his mattress when he was treated with gentamycin and prednisone. He was also treated with medications directed asthma. There was visible mold contamination found on the foam of his mattress in early 2013. The visible mold contamination was confirmed by testing. Once the mold contamination was discovered and he ceased having exposure to mold from the mattress, his state of health significantly improved. However, he continues to have loss of smell, profound hearing loss in the left ear, and bilateral cataracts.

His past medical history is negative for hypertension, diabetes, pulmonary disease, gastrointestinal disease, renal disease, endocrine disease, connective tissue disease, musculoskeletal disease, or disease of the nervous system.

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He denies any allergies. He had lightheadedness when he was prescribed tetracycline in the 1970s.

His sole medication is a proto-pump inhibitor for acid reflux as well as a daily baby aspirin.

He indicated that his mother died at the age of 89 due complications of a cerebral vascular accident. His father died at the age of 62 due to complications of a cerebral vascular accident.

He indicated that he has never been a smoker and only social alcohol. He works for the Lutheran Church for their investment services.

Review of systems is non-contributory.

PHYSICAL EXAMINATION

Constitutional Exam/General Appearance: The patient is a well-nourished man who is in no acute distress. Skin and Integuments: Normal skin turgor. H.E.E.N.T: Pupils are equal, round and reactive to light. Extraocular motions are intact. Cardiovascular Exam: Normal heart rate and rhythm without murmur or gallop. Pulmonary: Lungs are clear to auscultation bilaterally. Abdomen: Positive bowel sounds in all four quadrants. No palpable organomegaly. No rigidity or tenderness to palpation. Musculoskeletal: There are no obvious deformities of the musculoskeletal system. Extremities: There is no edema or cyanosis. Mental Status: Awake, alert, orientated. Cranial Nerves: I: Not tested. II: Normal overall vision. III, IV, VI Normal eye movements. V. Grossly normal mastication. VII: Normal and symmetrical facial movements. VIII: Hearing loss in left ear. IX. X: Normal swallowing function and reflexes. XI: Normal lateral neck movements. XII Normal tongue movement. Motor Examination: No motor deficits are noted. Sensory

Examination: No sensory deficits are noted. Coordination: Normal truncal and appendicular coordination. Gait: Normal ambulation without assistance. Neurovascular Exam: Normal pulses with capillary refill of less than 3 seconds.

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PEER REVIEWED LITERATURE

Bioaerosols: Assessment and Control is a 1999 publication of the American Conference of Governmental Industrial Hygienists (ACGIH). The ACGIH is a highly respected organization that provides guidance to the United States Government in the formulation of occupational and environmental exposure limits. In my opinion, this publication is a reliable authority and cites from this publication are reflective of recognized and generally accepted methodology by the medical and scientific community.

Bioaerosols: Assessment and Control indicates that the presence of visible mold is confirmation of mold contamination and that proper remediation includes correction of the conditions causing mold growth.

7.4.2 Fungi

Many fungi produce allergens and some fungi produce toxins. Fungal growth in buildings is undesirable and may cause health problems for building occupants. Although it may be difficult to establish that exposure to fungal aerosols occurs or that exposure presents a hazard, indoor fungal growth is inappropriate and should be removed. Further, steps should be taken to correct conditions that led to fungal growth so that it does not recur. Visible contamination that is confirmed by source sampling to be fungal growth is evidence of indoor contamination. Air sampling (Culture or spore-trap sampling) may also indicate indoor fungal growth but should be followed by inspection and source sampling to identify the location of fungal contamination.

Bioaerosols: Assessment and Control supports the assertion that allergic disease may be caused by mold antigen exposure after time is allowed for immunological sensitization.

25.2.2 Antigen-Mediated Diseases:

The pathogenesis of all hypersensitivity disease involves (a) repeated antigen exposure, (b) immunological sensitization of a host to an antigen, and (c) immune-mediated damage to the host. Time is required for the body to develop an immunological sensitization. Therefore, latency (absence of response or disease on first contact) is characteristic of all hypersensitivity diseases. The spectrum of disease associated with airborne antigen exposure may involve the upper or lower airways and

includes allergic asthma, allergic rhinitis, allergic sinusitis, atopic dermatitis (skin inflammation and itching), allergic mycosis (fungosis, most commonly allergic aspergillosis), and HP (hypersensitivity pneumonitis). The first three of these are allergic diseases that depend on the antigen-stimulated production of specific IgE antibodies.

Chronic sinusitis can cause olfactory disorders including loss of smell ***(Croy I, Nordin S, Hummel T. Olfactory Disorders and Quality of Life – an Updated Review. Chem. Senses 2014, 39:185-194. Litvack JR, Fong K, Mace J, James KE, Smith TL. Predictors of Olfactory Dysfunction in Patients with Chronic Rhinosinusitis. Laryngoscope. 2008. 118(12): 2225-2230)***.

Aminoglycosides including gentamycin are well known to cause ototoxicity with permanent hearing loss ***(Singer C, Smith C, Krieff D. Once-Daily Aminoglycoside Therapy: Potential Ototoxicity. Antimicrobial Agents and Chemotherapy. 1996. 40:9, 2209-2211. Cecil Medicine. 23rd Edition. © 2008. Saunders. ISBN 978-1-4160-2805-5. Page 2163.)***.

Steroids including prednisone are well known to cause cataracts ***(Furst C, Smiley WK, Ansell BM. Steroid Cataract. Ann. Rheum Dis. 1966 25; 364-368)***.

OPINION

Mr. Ralph Simon has suffered from disease in the form of allergic rhinitis and chronic sinusitis due to exposure to mold from his contaminated mattress. However, in addition, due to his treatment with gentamycin he has suffered permanent hearing loss. His treatment with gentamycin occurred due to his treatment of symptoms believed to be arising out of an infectious disease but were instead due to his mold exposure due to the contaminated mattress in this matter. He also was treated with steroids in the form of prednisone. The treatment with prednisone caused him to develop cataracts. The cataracts are permanent and may require future surgical treatment. The treatment with prednisone was due to his symptoms arise from his exposure to the hidden contamination of his mattress due to mold.

COMPENSATION: I have been paid a flat fee of $30,000 for all work involved in formulating my opinions and preparation of this report. Any additional services will be at the rate of $500 per hour for all services other than testimony time. Testimony time will be at the rate of $1,000 per hour.

CURRICULUM VITAE: My current curriculum vitae is attached to this report.

TESTIMONY LIST: The list of all my testimony for the last four years is attached to this report.

Very truly yours,

Ernest P. Chiodo, M.D., J.D., M.P.H., M.S., M.B.A., C.I.H.

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Olfactory Disorders and Quality of Life—An Updated Review

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Abstract

Olfactory disorders are common and affect about one-fifth of the general population. The main causes of olfactory loss are post viral upper respiratory infection, nasal/sinus disease, and head trauma and are therefore very frequent among patients in ear, nose, and throat clinics. We have systematically reviewed the impact of quantitative, qualitative, and congenital olfactory disorders on daily life domains as well as on general quality of life and depression. From the extensive body of literature, it can be concluded that loss of the sense of smell leads to disturbances in important areas, mainly in food enjoyment, detecting harmful food and smoke, and to some extent in social situations and working life. Most patients seem to deal well and manage those restrictions. However, a smaller proportion has considerable problems and expresses a noticeable reduction in general quality of life and enhanced depression. The impact of coping strategies is discussed.

Key words: Depression, olfaction, prevalence, quality of life, smell

For normosmic people, it is hard to imagine what life would be like without olfaction. Many physicians seem rather helpless in what to tell patients with olfactory disorders. The current review aims to give some guidance of what restrictions in daily life can be expected and how quality of life changes (QoL) in persons with olfactory disorders. We start with a brief overview about the function of olfaction, prevalence, and causes of olfactory disorders and continue with a detailed discussion about the consequences of olfactory disorders in domains where olfaction plays a major role. Furthermore, the impact of acquired quantitative, qualitative, and congenital olfactory disorders on general QoL as well as coping mechanisms is reported. A special section focuses on olfactory disorders among older people.

The role of olfaction

The general role of olfaction is to guide our attention towards hazards (e.g., microbial threats and poisonous fumes) and towards items with positive connotations (e.g., nutritious food). This guidance is predominantly driven by the valence (pleasantness/unpleasantness) of the odorous item (e.g., food), which—to a large extent—is determined by the individual's personal history with that item. To various degrees (see also Khan et al. 2007; Lapid et al. 2011; Kermen et al. 2013), odor preferences result from a learning process. At an earlier encounter with the food we associate its odor with a positive or negative emotion, and at the later occasion we recognize the odor and retrieve the association from memory (Engen 1991; Rolls 2004). Thus, the relatively strong positive or negative emotions often evoked by smells are shaped by prior experience and are believed to enhance the appropriate behavioral response.

Olfaction plays a major role in food intake, such that odors assist in food localization and indicate the food's edibility (Stevenson 2010). Important for food perception, the odorous molecules can also reach the epithelium from the oral cavity through the retronasal passage, which is enhanced by movements of the tongue, cheek, and throat that pump the molecules through this passage (Burdach and Doty 1987). A discrepancy between perceived flavor and expectation formed prior to ingestion can lead to rejection of the food. Several olfactory-related mechanisms are involved in appetite regulation by affecting our decisions on when, how much, and what to eat (Nordin 2009).

Olfaction may also be involved in social communication. Odors have been reported to have an impact on reproductive

behavior, including inbreeding avoidance and mate selection as well as emotional contagion (Stevenson 2010). The latter refers to the ability to detect fear-related cues (Ackerl et al. 2002; Prehn-Kristensen et al. 2009). Another example of this social function has been shown in a recent study in which female tears were demonstrated to contain chemical signals that decrease sexual arousal and testosterone levels in men (Gelstein et al. 2011).

Types of olfactory disorders and prevalence

Types of olfactory disorders.

Using appropriate tools (e.g., the "University of Pennsylvania Smell Identification Test," or the "Sniffin' Sticks"), quantitative smell disorders (anosmia, hyposmia) can be differentiated from normal olfactory function. Whereas most olfactory disorders are acquired, there are some patients who were born without a sense of smell, so-called congenital anosmia. In those patients, the olfactory bulb is typically hypoplastic or aplastic and accompanied by a shallow olfactory sulcus (Abolmaali et al. 2002). Some patients also exhibit qualitative olfactory disorders. Such disorders may be divided into parosmias and phantosmias—often characterized as unpleasant sensations (Leopold 2002). Parosmias are distorted odor perceptions in the presence of an odor source; phantosmias are odor percepts in the absence of an odor (Frasnelli et al. 2004). Phantosmias and parosmias are typically caused by classical causes of olfactory loss, for example, sinunasal disease, infections of the upper respiratory tract, or head trauma (Landis et al. 2005). Qualitative olfactory disorders seem to occur during states of neuronal degeneration or regeneration (Leopold 2002). However, phantom odors occur in psychiatric or neurological diseases; phantosmias may also occur in isolation as a single symptom (Pryse-Phillips 1971; Frasnelli et al. 2004; Frasnelli and Hummel 2005).

Prevalence of olfactory disorders.

Population-based studies of olfactory loss indicate a prevalence of 22% (25–75 years; Vennemann et al. 2008), 19% (≥20 years; Bramerson et al. 2004), or 24% (≥53 years; Murphy et al. 2002), with highest prevalence in older men. However, unawareness of olfactory loss is common (Nordin et al. 1995; Murphy et al. 2002; Shu et al. 2011) perhaps because olfactory information is processed unconsciously to a relatively large extent. Consequently, the prevalence of self-reported smell loss varies between 1.4% and 15% (Hoffman et al. 1998; Murphy et al. 2002; Nordin et al. 2004).

The most common etiologies of smell loss are post viral upper respiratory infection (URI) (18–45% of the clinical population) and nasal/sinus disease (7–56%) followed by head trauma (8–20%), exposure to toxins/drugs (2–6%), and congenital anosmia (0–4%) (Nordin and Bramerson 2008). A survey specific for Germany, Austria, and Switzerland shows similar results (Damm et al. 2004). Regarding how common smell loss is among different medical conditions, the percentage of patients with clinically proven smell loss is rather high: 76–95% in post viral URI, 72–98% in nasal/sinus disease, 86–94% in head trauma, 67% in exposure to toxins/drugs, and 100% in congenital cases (Nordin and Bramerson 2008). Loss due to post viral URI, head trauma, and exposure to toxins/drugs is to some degree reversible (Duncan and Seiden 1995; Reden et al. 2006), whereas many cases of nasal/sinus disease can be treated with medication or with a combination of conservative and surgical treatment (Seiden et al. 1992). Further information about the treatment of olfactory disorders can be found elsewhere (Hummel et al. 2011; Welge-Luessen and Hummel 2013).

The prevalence of congenital anosmia is estimated at 1:5000–10000 (Croy et al. 2012), and this disorder is frequently overlooked. We learned from our patients that approximately 13 years passed between the time when the disorder was noticed first (at about the age of 10 years) and the final diagnosis (Bojanowski et al. 2013).

For qualitative disorders, the prevalence is considerably lower than for quantitative disorders. In the general population, the prevalence of phantosmia is estimated between 0.8% and 2.1% (Landis et al. 2004) and parosmia to ~4% (Nordin et al. 2007). Among patients with olfactory disorders, parosmia may range from 10% to 60% (Deems et al. 1991; Nordin et al. 1996; Faulcon et al. 1999; Quint et al. 2001)—possibly indicating that the detection of parosmia is critically dependent on how the investigator asks for parosmia.

Consequences of olfactory disorders

Assessment of daily life problems in olfactory disorders and general QoL

Keller and Malaspina recently collected 1000 case reports giving very illustrative descriptions of daily life with olfactory disorders (Keller and Malaspina 2013). Several questionnaires have been developed to detect specific changes related to olfactory loss systematically (cf. Table 1 for overview). Results from those questionnaires form the base for the next chapter "Olfactory disorders and daily life functions."

Coping with olfactory disorders has been measured using a coping checklist (Lazarus and Folkman 1984). In addition, Nordin et al. (2011) introduced a questionnaire consisting of 5 problem-focused and 6 emotion-focused questions about coping with the olfactory loss.

General QoL can be assessed with questionnaires, such as the Short Form-36 Health Survey (Ware 2000), the General Well-Being Schedule (McDowell 2006), the 90-item Symptom Checklist (Derogatis 1977), the Mood Inventory (Zessen 1975), and the Nottingham Health Profile (Wiklund

Table 1 Questionnaires measuring changes related to olfactory loss

Reference	Description
Anderson et al. (1999)	Sinonasal Outcome Test-16
	Specifically addressing nasal dysfunction
Croy et al. (2010)	Importance of olfaction
	Addressing associations, applications, and consequences of olfaction in daily life
De Jong et al. (1999)	Appetite, hunger, subjective taste, and smell questionnaire Measuring those functions nowadays compared with past perception
Frasnelli and Hummel (2005); Neuland et al. (2011)	Questionnaire of Olfactory Disorders
	Assessing daily life problems associated with olfactory loss
Hufnagl et al. (2003)	Questionnaire for the assessment of self-reported olfactory functioning
Miwa et al. (2001)	Questionnaire on the impact of olfactory impairment on quality of life and disability
	Measuring impairment in 15 olfactory-related daily life activities and general enjoyment of life
Nordin et al. (2003)	Scandinavian adaptation of the Multi-Clinic Smell and Taste Questionnaire
	Assessing consequences of olfactory dysfunction
Pusswald et al. (2012)	Brief Self-Report Inventory to Measure Olfactory Dysfunction and Quality of Life
	Assessing the subjective general and odor specific olfactory function and olfaction related quality of life
Takebayashi et al. (2011)	Self-administered odor questionnaire
	Measuring how strong each of 20 odors are perceived
Varga et al. (2000)	Impact of chemosensory disorders on everyday life
	Assessing specifically olfactory-related changes in Quality of life

et al. 1988) (for overview see Bullinger 2002). Indications for depressive symptoms are often assessed with the Beck Depression Inventory (BDI) (Beck et al. 1961) or its more recent version (Beck et al. 1996). In using those measurements, one has to be aware that olfactory loss is often confounded with comorbidity. So, it is difficult to determine whether QoL reduction is due to the olfactory or the comorbid disorder. At least in patients with olfactory loss due to sinonasal disease, a major component of the decrease in QoL is related to decreased patency of the nasal airways, which severely contaminates the results obtained with these questionnaires. Chronic rhinosinusitis alone, for instance, has an impact on QoL, and cormorbidities, such as asthma and allergies, have cumulative negative effects (Alobid et al. 2008). One study in patients with chronic rhinosinusitis even indicates that the additional effect of olfactory loss on general QoL is negligible (Litvack et al. 2009). However, daily life restrictions from olfactory loss may easily be overseen, if not specifically asked for.

Olfactory disorders and daily life functions

Patients with olfactory disorders are impaired in areas of food intake, safety, personal hygiene, and in their sexual life (cf. Tennen et al. 1991; Van Toller 1999; Hummel and Nordin 2005) (Figure 1).

Most often, "difficulties related to eating" are examined and reported in patients with olfactory disorders. The perceived taste of food is strongly determined by olfactory experience, and a lack of the sense of smell consequently reduces the richness of food perception. A congenital anosmic women recently gave an illustrative example of this: "I had many lunchtime meetings with academics who drank wine enthusiastically and enjoyed talking about it. I listened to them carefully and also read the labels on the bottle, which are a great literary genre, with fascinating poetical descriptions of taste, and I imagined the vineyards in the sunshine and old oak barrels in cellars. But when I drank, I only noticed a bitter sensation that was not particularly pleasant." (Tafalla 2013).

Ferris and Duffy found that 69% of their patients ($n = 239$) enjoyed food less than before onset of the disorder (Ferris and Duffy 1989) (see also Varga et al. 2000; Hufnagl et al. 2003; Keller and Malaspina 2013). The reduced experience of food quality led to diminished appetite in 27% of their patients. Reduced appetite was also stated by 27% ($n = 50$) (Nordin et al. 2011), 32% ($n = 72$) (Blomqvist et al. 2004), and even 56% ($n = 278$) (Temmel et al. 2002) of patients. The actual eating behavior seems to depend on coping mechanisms. Patients report to increase the taste by using more salt, sweetener, or irritants/spices as well as valuing the texture more. One patient reported, "I ended up gaining almost twenty pounds before realizing I was consuming more of every food in an effort to taste it"(Keller and Malaspina 2013). Studies show that the percentage of patients reporting to eat more varies between 3% and 20%, and between 20% and 36% report to eat less since the onset of the olfactory

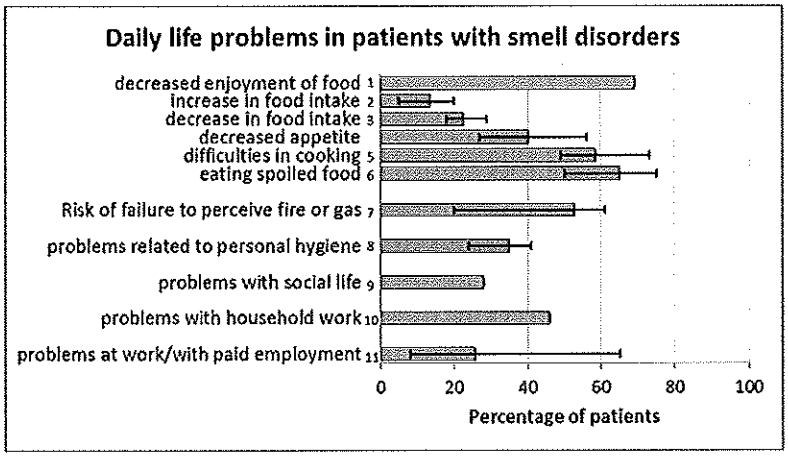

Figure 1 Daily life problems in patients with smell disorders. Results of 8 studies are included. The bars visualize the mean percentage of patients affected, weighted by the number of participants per study. The error bars show the lowest and highest reported percentage in the studies. The calculation is based on the following studies: Temmel et al. (2002), $n = 278$ (items 4–6, 8, 11); Tennen et al. (1991), $n = 66$ (item 8); Miwa et al. (2001), $n = 420$ (items 5–8); Nordin et al. (2011), $n = 50$ (item 4); Ferris and Duffy (1989), $n = 230$ (item 1–4); Brämerson et al. (2007), $n = 102$, (items 9–11); Blomqvist et al. (2004), $n = 72$ (item 4); Aschenbrenner et al. (2008), $n = 176$ (items 2,3).

disorder (Ferris and Duffy 1989; Aschenbrenner et al. 2008; Keller and Malaspina 2013). The numbers imply that a large percentage of these patients have problems in maintaining their original eating behavior. However, it is rather unclear why some patients keep their weight, whereas others eat either less or more after food have become "tasteless." Coping mechanisms like eating after time schemes and enriching food by other sensory information, such as texture and color, are often reported to be helpful.

It is unclear whether patients with parosmia or phantosmia are more affected than those with a quantitative disorder. In the study of Aschenbrenner et al. (2008), food consumption was not different between patients with quantitative versus qualitative olfactory disorders. On the other hand, patients with parosmia and phantosmia are reported to exhibit specific dislikes towards foods (Mattes et al. 1990), and single-case reports relate phantosmia to severe weight loss (e.g., Muller et al. 2006). No significant weight difference, and no difference in food preferences, was found in patients who were born without the sense of smell in comparison to an age-matched control group (Croy et al. 2012). This is also supported from observations, indicating that congenital absence of olfaction does not result in markedly aberrant food preferences (Doty 1977).

Food-related problems are not limited to eating; preparation of food is difficult for many patients with olfactory disorders. Problems with cooking have been reported in 49% ($n = 420$) (Miwa et al. 2001) and 73% (Temmel et al. 2002) of the patients. In those studies, half and two-thirds of the patients, respectively, had problems detecting spoiled food (Miwa et al. 2001; Temmel et al. 2002). Santos et al. (2004) asked about hazardous events: 37% of their patients (total $n = 445$) reported at least one such event, and cooking-related hazards were reported most often followed by detecting spoiled food. Despite having acquired better coping mechanisms (Bojanowski et al. 2013), congenital anosmic persons also report enhanced problems with detecting burning food and spoiled food (Croy et al. 2012).

Another common problem is the "failure to detect fire, gas or smoke", which is reported by 61% of the patients (Miwa et al. 2001). A congenital anosmic women, for instance, told us, "My husband was a policeman who worked a lot of night shifts, leaving me alone at night with the children. Until my oldest children were old enough to be reliable, I worried constantly about gas leaks and fire in the night when I wouldn't be able to detect them until it would be too late. Once my older children got about 8–10 years old and understood the importance the lack of smell made to our safety at night, I felt much safer." Twenty percent of the smell patients describe not having been able to detect fire related smoke (Haxel,et al. 2012) and even more worry about potentially not detecting such a danger. The failure to detect fire or smoke was described as the main risk associated with olfactory disorders by 38 to 45% of the patients (Blomqvist et al. 2004; Nordin et al. 2011).

Furthermore, patients typically express problems related to "personal hygiene". They worry about their body odor, bad breath, and their children's hygiene: "My 8 children all learned very early to tell me when their diapers needed changing, and another way I coped with that was to check them almost constantly."

Worry about not being able to perceive the own body odor was reported by 41% of the patients of Temmel et al. (2002). In 2 studies, 19% and 36% of the patients described being less aware of personal hygiene as the most negative effect of

the olfactory disorder (Blomqvist et al. 2004; Nordin et al. 2011). In line with this, 33% of the patients of Miwa et al. (2001) stated problems in using perfume.

Maybe related to insecurity about personal body odor, "social relations" are reported to be affected by olfactory disorders. One-fourth to one-third (Tennen et al. 1991) of the patients report such problems (Varga et al. 2000; Bramerson et al. 2007; Nordin et al. 2011). Impaired sex life has been reported by Hufnagl et al. (2003), but Brämerson et al. (2007) found no enhanced sexual problems compared with a control group. Depression and gender might be moderating factors. In the study of Gudziol et al. (2009), patients reported small but significantly reduced sexual appetite after, compared with before, the onset of the smell disorder. This was more pronounced in men with more severe depression. The same gender effect was found in congenital anosmic patients. Men born without a sense of smell described a reduced number of sexual relationships (Croy et al. 2013).

Problems in "working life" have been reported by 8% (Temmel et al. 2002) up to about one-third of the patients (Bramerson et al. 2007), depending on the question asked. Whether olfactory impairment interferes with working life depends on profession. Reduced ability in working was described as the main interference of olfactory disorder in daily life by 3–8% of the patients (Blomqvist et al. 2004; Nordin et al. 2011). For persons working as cooks or wine tasters, perfumers, nurses or firemen, olfactory disorders can be catastrophic. Haxel et al. (2012) asked their patients specifically about consequences for working life. Only 35% of their patients continued working without restrictions, 60% of their patients needed special adjustments on their job, and 5% of the patients reported not being able to work anymore in their former profession.

General QoL and Depression

Olfactory disorders impair QoL, and we propose 2 potential pathways (see Figure 2). First, it is reasonable to assume that the aforementioned daily life restrictions impair QoL and enhance the likelihood of depression. Reduced food enjoyment and social security as well as worries about personal hygiene may reduce participation in social life and make persons more prone to depression. Worries about the professional future may add to this. Secondly, it is likely that olfactory loss per se affects the brain's functioning and, especially, emotional control. A potential mechanism is the reduced input from the olfactory bulb via amygdala into the limbic circuit (cf. Leonard 1984; Kelly et al. 1997; Masini et al. 2004; Song and Leonard 2005, for studies on rodents, and Deems et al. 1991; Temmel et al. 2002; Pause et al. 2003; Croy et al. 2011; Landis et al. 2012, for studies on humans).

About one-fourth to one-third of patients with smell disorders exhibit depressive symptoms. Deems et al. (1991) found that 25% of their 750 patients reported BDI scores in the range of mild to severe depression. Among the patients with accompanying parosmia or phantosmia, 35% exhibited high depression scores. Similarly, Tennen et al. (1991) showed that a feeling of vulnerability was the single most stressful aspect of an olfactory disorder, as reported by 28% of 196 investigated patients. BDI scores indicating moderate depression were found in 17% of their patients. Miwa et al. (2001) report that 25% of their patients enjoyed life less than before the disorder onset. A similar high proportion of reduced QoL was reported in 2 other studies: 27% and 30% of the patients of Blomqvist et al. (2004) and Nordin et al. (2011), respectively, indicated severe distress in the General Well Being Questionnaire. This might be related to coping. Twenty-seven percent of the patients of Nordin et al. (2011) did not agree to the statement "do you accept the situation and try to make the best out of it." Among 32 patients, who were born without the sense of smell, 29% report BDI scores in the range of mild to severe depression (Croy et al. 2012).

Reduced QoL in olfactory-specific domains and in general health was also reported in a study ($n = 205$) by Frasnelli and Hummel (2005). Importantly, QoL was reduced even more in parosmia/phantosmia patients. Also, Bonfils et al. (2005) found that half of their 56 patients with parosmia and moderate to severe olfactory loss reported severely affected QoL.

In a study in people with self-reported loss of smell ($n = 90$), enhanced depression and reduced QoL based on the SF-36 was found in over 20% of the respondents (Smeets et al. 2009). A very high proportion, namely 68%, of the patients of Temmel et al. (2002) have been reported to exhibit signs of depressed mood assessed with a single item only—which might explain the difference to other studies. However, the authors found that those patients with signs of depressed mood had significantly higher complaints about olfactory-related daily life problems.

Coping with the olfactory deficit

The majority of patients develop strategies to adjust to the olfactory disorder. Patients with parosmia and phantosmia have increased problems in coping compared with patients with only quantitative disorders (Frasnelli and Hummel 2005). Tennen et al. (1991) analyzed different coping strategies in patients and came to the conclusion that the strategy used to cope with the olfactory loss and the appraisal of the loss contributes to psychological well-being. Problem- and emotion-focused coping strategies are applied by about 80% of the patients (Nordin et al. 2011). For instance, trying to accept the situation and making the best out of it is the emotional coping strategy used by most of the patients. Asking family members for support in tasting food are problem-focused strategies used by a similar high proportion of about two-thirds of the patients (Blomqvist et al. 2004). Another coping mechanism frequently reported is the purchase of gas and smoke detectors.

In a study including 235 patients, we found adjustment to impaired olfactory function by giving this domain less

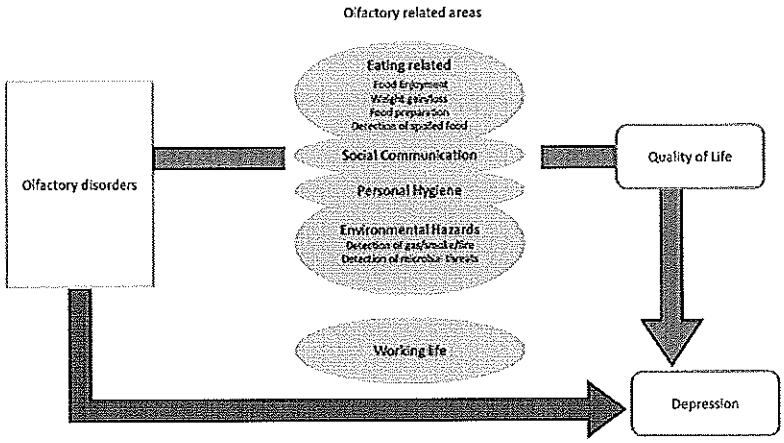

Figure 2 Pathways of depression in olfactory disorders. Olfactory impairment leads to restrictions in olfactory-related areas, which can affect Quality of life and, by this pathway, enhance depression likelihood. Working life is directly affected in professions depending on olfactory ability, such as perfumers, firemen, or cooks. However, working life is also impaired in professions where olfactory-related areas play a major role, such as detecting microbial threats in nurses. A second pathway refers to potentially altered brain functioning in olfactory disorders.

importance (Croy et al. 2011). Compared with hyposmic patients, anosmic patients stated that they try to use the sense of smell less often in daily life. Both groups rated their sense of smell as less important than a group of normosmic people. Interestingly, 13% of the patients expressed enhanced scores in an aggravation scale (e.g. "without the sense of smell my life would be worthless"), indicating that they exhibit major problems adjusting to the deficit. These patients also exhibited significantly higher depression scores. However, there seems to be only a small number of patients with major problems in coping with the impairment. That is probably why Frasnelli and Hummel (2005) found no general correlation between coping and depression.

Severity and duration of the olfactory impairment influencing QoL

In a study conducted by Simopoulos et al. (2012), a very high correlation ($r = -0.7$) was found between olfactory dysfunction and olfactory-related QoL in a group of 102 chronic rhinosinusitis patients with and without olfactory deficits. Furthermore, the more pronounced the olfactory disorder, the more symptoms of anxiety and depression were reported. However, inclusion of a group without impaired olfactory function is likely to overestimate the coherence. Among groups of patients with olfactory loss only, the correlation between olfactory impairment and olfactory-related QoL (Frasnelli and Hummel 2005; Neuland et al. 2011) and general QoL was rather low (Neuland et al. 2011). There are indications that general QoL is reduced more severely in hyposmic compared with anosmic patients (Neuland et al. 2011). The authors interpret this as enhanced hope for recovery in hyposmic patients, which may prevent attempts to cope with the disorder.

When asked specifically about several domains related to olfaction, disease duration showed no influence on daily life disturbance (Temmel et al. 2002; Neuland et al. 2011). However, adjustment over time can be seen when patients are asked in a different way. Decreased enjoyment of food is less pronounced when the disorder lasts more than 3 years (Ferris and Duffy 1989) and the number of household hazards decreases over the first 2 years (Bojanowski et al. 2013). Along the same line, Tennen et al. (1991) report that patients with longer disorder duration exhibit lower scores in the BDI . In accordance, patients with a disorder duration of more than 1 year tended to use their sense of olfaction less often than patients with shorter disorder duration, indicating adjustment (Croy et al. 2011). Shu et al. (2011) found a positive correlation between disorder duration and score on the Positive Statements subscale of the Questionnaire of Olfactory Disorders among their 413 patients, indicating that patients learn to cope with their olfactory loss.

Influence of age

Physiological anorexia is common in the older population and may—at least to some degree—be explained by olfactory loss, which also means loss of retronasal olfactory function affecting flavor perception. Data from older people with changes in olfactory perception also suggest a decrease in food appreciation and appetite, change in food choice such as decreased dietary variation, poor nutritional status, change in body weight, and an increased risk for chronic disease (Fanelli and Stevenhagen 1985; Wysocki and Pelchat 1993; Mattes and Cowart 1994; Duffy et al. 1995; Griep et al. 1995; Morley 2001; Wilson and Morley 2003; Karpa et al. 2010). Rolls and McDermott have demonstrated that sensory-specific satiety is less pronounced in older people compared with young adults, which may explain the decreased dietary

variation with age (Rolls and McDermott 1991). However, not all studies have shown a relation between chemosensory impairment and nutritional problems (Ferris and Duffy 1989).

There may also be a considerable risk among older people to ingest spoiled food. It has, for example, been suggested that older adults are less likely than young adults to reject foods with unpleasant odors (Pelchat 2000). It is possible that this results in increased risk of minor gastrointestinal complaints, which is a common condition among elderly (Firth and Prather 2002). Importantly, Schiffman and collaborators have reported that anorexia in the older people often remits when foods are amplified by additional flavoring (e.g., artificial chicken flavor on a chicken dish) to compensate for diminished chemosensory function (Schiffman and Warwick 1988). More specifically, additional flavoring seems to increase institutionalized older peoples' preference for and intake of food (Schiffman 1998), increase salivation (Schiffman 1998; Schiffman and Miletic 1999), and improve immunological status and grip strength (Schiffman and Warwick 1993). However, more recently, these early findings have been discussed controversially (Koskinen et al. 2005; Kremer et al. 2007).

Boesveldt et al. (2011) observed a small correlation between olfactory function and depression in older persons. In a line, Seo et al. (2009) reported that olfactory disorders were significantly associated with low QoL and depression in older people and also with low cognitive function. However, the associations with QoL and depression did not remain when controlling for cognitive function. This illustrates the importance of controlling for incipient dementia when studying QoL and depression in older people with olfactory impairment. Olfactory impairment is an early sign of Alzheimer's disease (Nordin 2012) and Parkinson's disease (Ponsen et al. 2004), and poor QoL and depression are common in dementia (Hoe et al. 2006). Nevertheless, associations between olfactory impairment and poor QoL and depression have been reported even after controlling for loss in cognitive function among older people and are associated also with functional disability and reduced independence (Gopinath et al. 2012).

Final remarks

Olfaction plays an important role for ingestion, harm avoidance, and social communication. However, about one-fifth of the population exhibits smell disorders, and most of them are not aware of it. Those persons who seek medical treatment often have problems finding a physician who is familiar with smell disorders (Haxel et al. 2012). Almost exclusively, patients presenting to such specialized physicians or to smell and taste centers are included in studies about the consequences of olfactory loss. This means that the basic population of people with smell disorders is not represented equally but that there is a strong bias towards patients with a certain psychological strain. This has to be kept in mind when we conclude that olfactory loss leads to disturbances in olfactory important areas, mainly in eating, detecting of harmful food and smoke, and to some extend, in social situations and working life. Most of the patients seem to cope well with these restrictions. However, about one-third of the patients with acquired and congenital olfactory disorders have more severe problems and express a noticeable reduction in QoL.

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Predictors of Olfactory Dysfunction in Patients with Chronic Rhinosinusitis

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Abstract

Objectives—To measure the prevalence of and identify clinical characteristics associated with poor olfactory function in a large cohort of patients with chronic rhinosinusitis (CRS).

Study Design—Multi-institutional, cross sectional analysis.

Methods—An objective measure of olfactory dysfunction, the Smell Identification Test (SIT), demographic data, clinical factors and co-morbidity data were collected from a cohort of 367 patients who presented with CRS at three tertiary care centers. Data was analyzed using univariate and multivariate analyses.

Results—Sixty-four percent of men and women aged 18 to 64 had olfactory dysfunction whereas 95% of patients ≥ 65 years had olfactory dysfunction ($p<0.001$); no significant difference was noted by gender. By multivariate logistic regression analysis, patients with nasal polyposis (OR 2.4, 95% confidence interval (CI) 1.3, 4.2; $p=0.003$) and patients ≥ 65 years (OR 10.0, 95% CI 2.3, 43.7; $p=0.002$) were at increased risk of hyposmia. Patients with nasal polyposis (OR 13.2, 95% CI 5.7, 30.7; $p<0.001$), asthma (OR 4.2, 95% CI 1.8, 9.8; $p=0.001$), ≥ 65 years (OR 15.6, 95% CI 2.3, 104.9; $p=0.005$), and smokers (OR 7.6, 95% CI 1.8, 31.6; $p=0.005$) were at increased risk of anosmia.

Conclusions—Poor olfactory function is common in patients with CRS. Age, nasal polyposis, smoking, and asthma were significantly associated with olfactory dysfunction in patients with CRS. Neither prior endoscopic sinus surgery nor a history of allergic rhinitis was associated with olfactory dysfunction. Septal deviation and inferior turbinate hypertrophy were associated with normal olfactory function.

Keywords

predictors; chronic rhinosinusitis; olfactory dysfunction; anosmia; hyposmia

INTRODUCTION

Chronic rhinosinusitis (CRS) is a common cause of olfactory dysfunction accounting for 14-30% of cases and affecting more than 10 million people.[1-3] The etiology of olfactory dysfunction in CRS is not well understood but appears to be multifactorial and involve both

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conductive and sensorineural mechanisms.[3] Mechanical obstruction by nasal polyposis and respiratory mucosal edema can lead to decreased airflow to the olfactory cleft and a conductive disorder. Direct inflammation of the olfactory neuroepithelium in histopathology studies and clinical response to systemic corticosteroids support a sensorineural component to the disorder.[4,5]

Despite extensive advances in the understanding of the basic science of olfaction, advances in the management of clinical olfactory dysfunction have been limited.[6] Some clinical factors, age, gender, exposure to toxic agents, various disease states and smoking, have been extensively studied and shown to be associated with olfactory dysfunction.[7-9] However, the role of common comorbidities in the relationship between olfactory function and CRS has not been well studied. Nasal polyposis, allergic rhinitis, asthma, septal deviation, inferior turbinate hypertrophy have been proposed to cause olfactory dysfunction, but the data is conflicting.[7, 10-13] Prior sinus surgery has also been reported as a cause of olfactory dysfunction.[12] Furthermore, the olfactory outcomes after medical and surgical treatment for CRS have been mixed.[7]

In this large, multi-institutional cross-sectional analysis, we examine the prevalence of and clinical characteristics associated with poor olfactory function in patients with CRS. We hypothesize that age, gender, nasal polyposis, asthma, and smoking predict poor olfactory function whereas a history of sinus surgery and/or a history of allergic rhinitis do not. In turn, findings from this study may help to inform clinicians which patients are at highest risk for olfactory dysfunction and in need of patient safety counseling as well as further treatment.

MATERIALS AND METHODS

Study Population and Data Collection

Study subjects were recruited from three tertiary care centers over a three-year period (n=396). All patients had a diagnosis of CRS based on the Rhinosinusitis Task Force criteria and endorsed by the American Academy of Otolaryngology — Head and Neck Surgery.[14] Adult (≥18 yrs old) patients were enrolled at the time they had failed maximum medical management and had elected to undergo sinus surgery. Subjects completed a medical history intake form and underwent a physical exam. Demographic and comorbidity data were documented by the physician during the interview and confirmed by physical exam and nasal endoscopy where appropriate. Variables included: age, gender, current tobacco use, prior sinus surgery, nasal polyposis, asthma, allergic rhinitis confirmed by allergy testing, acetylsalicylic acid (ASA) intolerance, septal deviation and inferior turbinate hypertrophy. Allergy testing was dependent on the clinical circumstances and may have included skin prick testing, modified radioallergic sorbent system (mRAST), or both. Patients < 18 years old, with immunodeficiency (n=5), autoimmune disease (n=8), and/or cystic fibrosis (n=12) were excluded from the study. Patients with olfactory test scores consistent with malingering (score ≤ 5) (n=4) were also excluded. A total of 367 patients (93%) were available for analysis. All study protocols and informed consent were collected and approved by Institutional Review Boards at each study site. All data was collected prospectively.

Measurement of Olfactory Function

The Smell Identification Test (SIT) from Sensonics, Inc., an objective measure of olfactory dysfunction with robust age and gender-specific normative data, was administered to patients by a trained research coordinator.[15] The SIT is a validated 40 question forced-choice test (total score: 0-40). It has high test-retest reliability ($r > 0.90$) and is highly correlated with more sophisticated measures of olfactory dysfunction ($r > 0.80$).[16-18] Absolute SIT scores are categorized into olfactory severity categories based on gender-specific normative data.[15] Men

with SIT scores ranging from 34-40 and women with SIT scores ranging from 35-40 are categorized as normosmic. Men with SIT scores ranging from 19-33 and women with SIT scores ranging from 19-34 are categorized as microsmic or hyposmic. Men and women with SIT scores ranging from 6-18 are categorized as anosmic. SIT scores ranging from 0-5 are consistent with malingering. SIT scores also vary with age; however, age is not incorporated into the olfactory severity classification system. To account for this, age was included as a covariate and adjusted for in the analysis.

Statistical Analyses

Analyses were conducted using SPSS v15.0 statistical software (SPSS Inc., Chicago, IL). Demographic and clinical characteristics of study subjects were analyzed by olfactory severity category. The prevalence of normosmia, hyposmia, and anosmia was calculated for age groups (18-39 years, 40-64 years, 65+ years). Differences across the three olfactory categories by age grouping were assessed using the Jonckheere-Terpstra test for doubly ordered categories.[19] Differences in the demographic data and clinical characteristics were assessed across the three olfactory severity categories. A one-way analysis of variance (ANOVA) was used to determine differences in the mean ages for the three olfactory categories. The Kruskal-Wallis test for ordered categories was used to determine differences across the olfactory categories for gender and clinical characteristics.[20]

Two multivariate logistic regression models were constructed to examine the relationship between clinical characteristics of patients with CRS and olfactory dysfunction. The first model examined which variables were associated with hyposmia and the second model examined which variables were associated with anosmia. Age was dichotomized into two groups. This decision was made based on inspection of normative data which showed that the median SIT score for patients remained stable across early and middle adulthood and then dropped precipitously in late adulthood.[15] The variable was divided into patients aged ≥ 65 years old and patients aged < 65 years old to account for this curvilinear relationship between age and normative olfactory data. All other independent variables were also dichotomized. Preliminary multilogistic regression models were constructed to include variables with univariate significance at $p < 0.25$ level. Final models were selected using forward selection ($p < 0.05$) and backwards elimination ($p > 0.10$) procedures in a stepwise fashion. Clinical factors known to predict olfactory dysfunction, such as age, gender, and smoking, and other clinical factors that were hypothesized to cause olfactory dysfunction, such as allergic rhinitis and turbinate hypertrophy, were forced into the model, but allowed to fall out if they did not remain significant. Odds ratios and 95% confidence intervals were reported. A linear regression model with the absolute SIT score as the dependent variable was also examined with similar results.

RESULTS

Thirty-three percent of patients presented with normosmia (n=122), 45% with hyposmia (n=166), and 22% with anosmia (n=79). The demographic and clinical characteristics of normosmic, hyposmic, and anosmic patients are described in Table I. The mean age of patients with normosmia was 43.8 years (± standard deviation (SD) 12.5) whereas it was significantly older in hyposmic (49.8 years (± SD 13.6)) and anosmic (49.4 yrs (±SD 13.8) patients ($p<0.001$). Nasal polyposis, asthma, and aspirin intolerance were more common in patients with olfactory dysfunction (all $p<0.001$). Olfactory dysfunction was also more common in patients undergoing revision surgery ($p=0.026$). Septal deviation was more common in patients with normal olfactory function or less severe dysfunction ($p<0.001$); turbinate hypertrophy trended in a similar direction although this did not meet statistical significance ($p=0.077$). There were no statistically significant differences in gender, ethnicity, the proportion of smokers or the proportion of allergic patients by group.

The prevalence of hyposmia and anosmia by age and gender are illustrated in Figure I and Figure II. The proportion of patients with olfactory dysfunction was similar between the ages of 18-39 years and 40-64 years for men (60.5% vs 65.6%, p= 0.550) and women (62.7% vs 64.1%, p= 0.860). Ninety-five percent of men and women greater than or equal to 65 years old had olfactory dysfunction; this was significantly greater than patients under 65 years old (p=0.008 and p=0.007, respectively).

Patients with nasal polyposis (OR 2.38, 95% CI: 1.34, 4.23, p=0.003) and age ≥ 65 years (OR 10.0, 95% CI: 2.30, 43.71, p=0.002) were at significantly increased odds of hyposmia as compared to patients < 65 years old and non-polyp patients (Table II). Patients aged 65 years or greater (OR 15.59, 95% CI: 2.32, 104.86, p=0.005), with nasal polyposis (OR 13.21, 95% CI: 5.68, 30.70, p≤0.001), asthma (OR 4.21, 95% CI: 1.81, 9.81, p=0.001), and/or current smokers (OR 7.58, 95% CI: 1.82, 31.55, p=0.005) were at increased odds of anosmia as compared to patients under 65 years, without nasal polyposis, non-asthmatics and non-smokers (Table II). Patients with inferior turbinate hypertrophy (OR 0.10, 95% CI: 0.01, 0.91, p=0.041) and/or septal deviation (OR 0.19, 95% CI: 0.06, 0.60, p=0.005) were less likely to have anosmia. Patients who had ASA intolerance, allergic rhinitis and/or prior sinus surgery were at neither increased nor decreased risk of olfactory dysfunction.

DISCUSSION

Clinical characteristics associated with olfactory dysfunction were examined at the time patients with CRS enrolled in a large multi-institutional, prospective cohort study. Two logistic regression models were designed to look at clinical predictors of hyposmia and anosmia as compared to patients with normosmia. We found that age ≥ 65 years, nasal polyposis, asthma, and smoking were associated with olfactory dysfunction in patients with CRS. Interestingly, when these variables were accounted for in multivariate logistic regression modeling, aspirin intolerance, allergic rhinitis, and history of prior sinus surgery were not significantly associated with olfactory dysfunction. Gender was adjusted for in the SIT scoring system based on normative data and did not require further adjustment as an independent variable in the model. Furthermore, there was no evidence of an interaction, or effect modification, between gender and CRS. Additionally, patients with septal deviation and inferior turbinate hypertrophy were less likely to have olfactory dysfunction than other patients with CRS.

Both linear and logistic regression models were examined with similar results. By reporting the results in terms of the logistic regression models, we were able to translate the associations between clinical indicators and olfactory dysfunction into quantifiable entities. Additionally, it allowed us to adjust for the nonlinear relationship between age and olfactory dysfunction.

As evident in this study and in the literature, the relationship between CRS and olfactory dysfunction appears to be multifactorial and complex. Traditionally, CRS was thought to be primarily a conductive disorder that caused mechanical obstruction of the olfactory cleft from physical obstruction of the nasal airways, nasal polyposis, edema, and secretions. However, causes other than nasal airflow appear to contribute to olfactory dysfunction.[7] Kern noted that patients with CRS have evidence of direct inflammation of the neuroepithelium and that the degree of inflammatory changes in the neuroepithelium was related to the severity of olfactory dysfunction.[5] This, as well as partial responses to systemic corticosteroids, has led to a better understanding that olfactory dysfunction by CRS is both a conductive and sensorineural process. Despite extensive knowledge regarding the underlying mechanisms of olfactory dysfunction, advances in the management of clinical olfactory dysfunction have been limited and treatment with oral steroids and surgery have demonstrated mixed results.

Several clinical cofactors were noted to impact the relationship between CRS and olfactory dysfunction, including age ≥ 65 years, nasal polyposis, asthma and smoking. Age is a known risk factor for olfactory dysfunction.[9] There is increased pro-apoptotic gene expression in the olfactory mucosa of older rats and increased olfactory receptor neuron cell death.[21-22] Additionally, patients with CRS and anosmia manifest moderate to severe inflammatory changes in the olfactory mucosa; inflammation, in turn, may inhibit olfactory neurogenesis.[5] Consequently, one wonders if CRS and age may have a synergistic effect on fragile neuroepithelium in a "two-hit" fashion where older patients are more susceptible to olfactory neuroepithelial injury and less able to recover from injury.

Smoking was also associated with olfactory loss. Other studies have shown that olfactory function is inversely related to cumulative dose of smoking as well as time since last cigarette.[8] Frye, et al showed that current smokers are 1.9 times (95% CI: 1.0, 3.8) as likely to have olfactory dysfunction as never smokers. The magnitude of this effect is relatively modest in that a four-point difference in SIT scores is noted between the highest and lowest risk groups. We found that current smokers are more likely to be anosmic than nonsmokers. As we did not measure quantity of cigarettes or time since last cigarette, we may have biased our results towards the null hypothesis.

In our model, nasal polyposis was associated with hyposmia and anosmia. Patients with nasal polyposis may suffer from a conductive olfactory loss caused by physical obstruction of the airway; they may also experience degenerative changes associated with recurrent infections, scarring, and chronic nasal medication use from long-standing polyposis.[23] Our findings are consistent with other studies.[23-25] Perry et al reported that patients with CRS and comorbid nasal polyposis had higher subjective olfactory dysfunction scores than non-polyp patients.[24] Vento found that 46% of patients with long-standing nasal polyposis had elevated olfactory thresholds above the upper limit of the 95% confidence interval for a similar aged reference group.[23] A population-based study of 1387 Swedish adults found that individuals with nasal polyposis were at increased odds of olfactory dysfunction (OR 2.1, 95% CI: 1.0, 4.3) and anosmia (OR 3.8, 95% CI: 1.6, 8.8) after adjustment for other clinical characteristics, including age and gender.[25]

In this study, patients with comorbid asthma were more likely to have olfactory dysfunction. Patients with CRS and asthma suffer from systemic inflammatory responses of the upper as well as the lower airway.[26] It appears that this systemic inflammatory process may affect the olfactory cleft as well. Our findings are consistent with subjective reports that olfactory dysfunction is a common complaint in patients with CRS and comorbid asthma.[24,27]

In contrast, allergic rhinitis was not associated with olfactory dysfunction in our patient population. Allergic rhinitis often manifests itself in high flow areas of the nasal airway, such as the inferior turbinate and middle meatus, where exposure to environmental toxins in maximized; our findings suggest that lower flow areas, such as the olfactory cleft, are less affected. Apter, et al has argued that patients with allergic rhinitis are at increased risk of olfactory dysfunction as a result of recurrent respiratory tract infections that lead to damage to the olfactory neuroepithelium.[10] In his study, allergic rhinitis patients with co-morbid CRS or nasal polyposis had worse olfactory function than patients with allergic rhinitis alone in a Taste and Smell Clinic. However, the subset of patients who presented to an allergy clinic for issues unrelated to olfactory complaints had normosmic olfactory thresholds. Our results are consistent with Simola, et al who found that nonallergic rhinitis patients had a poorer sense of smell than patients with seasonal or perennial allergic rhinitis.[11]

A history of prior sinus surgery was not associated with olfactory dysfunction. Several mechanisms have been proposed to cause olfactory injury after sinonasal surgery: direct injury

to the olfactory epithelium, modification of airflow, effects of pharmacologic agents, scarring, and/or vascular injury and ischemia.[12,28] A 1% risk of anosmia following nasal surgery has been reported.[12] However, we found that patients who had previously undergone ESS were not at increased risk for olfactory dysfunction compared to patients who had no history of sinus surgery. This is particularly interesting because according to the descriptive data, a higher proportion of patients with a history of ESS had olfactory dysfunction. However, when other variables were adjusted for in the multivariate logistic regression model, it became evident that these other variables, namely age, nasal polyposis, asthma, smoking, not a history of prior surgery, accounted for olfactory dysfunction. A previous study noted that a history of surgery for nasal polyposis was associated with poor olfactory function but a history of surgery for chronic maxillary sinusitis was not.[11] We suspect that it was the underlying medical comorbidity, nasal polyposis, that contributed to the olfactory dysfunction rather than the surgery. By accounting for both variables, nasal polyposis and prior sinus surgery, in our multivariate logistic regression model, we found that it was the presence of nasal polyposis not the history of prior surgery that was associated with olfactory dysfunction. Consistent with our findings, Vento et al also noted that there was no relationship between number of surgeries and olfactory threshold in patients with nasal polyposis.[23]

Olfactory dysfunction is common in aspirin intolerant patients. The mean SIT score in aspirin intolerant patients was 20.7 (± SD 10.9), but the variable was not significant in the logistic regression model. We suspect this is a limitation of the data. The power to analyze the variable may be limited by the relatively small number of aspirin intolerant patients in the study. Furthermore, both asthma and nasal polyposis were adjusted for in the model and there may be an aspect of colinearity that makes aspirin intolerance difficult to analyze. In our clinical experience, patients with aspirin intolerance frequently suffer from olfactory dysfunction although we were unable to detect it.

Septal deviation and inferior turbinate hypertrophy have been hypothesized to cause olfactory dysfunction via physical obstruction of the nasal airway.[13] In this cohort of patients with CRS, septal deviation and inferior turbinate hypertrophy were negatively associated with olfactory dysfunction. As both diagnoses were determined by the clinician, they are subjective in nature and susceptible to potential bias. Similar to this study, Kimmelman et al found that patients undergoing septoplasty had normal preoperative SIT scores (mean SIT score 34.0) as compared to patients undergoing ethmoidectomy (mean SIT score 21.8) or polypectomy (mean SIT score 17.1).[12] Studies have shown mixed results regarding olfactory function after surgical treatment of nasal obstruction caused by septal deviation and/or inferior turbinate hypertrophy. In Kimmelman's study, there was no statistically significant difference in post-operative mean SIT scores of patients undergoing septoplasty, although one patient became anosmic.[12] In contrast, Damm et al showed that the majority of patients reported improved odor identification (80%) and odor discrimination (70%).[13] Surgical treatment of septal deviation and/or inferior turbinate hypertrophy may alleviate nasal obstruction in this population but the impact on olfactory function is less evident. Future data collection on our patients post-operatively will help to further delineate this relationship.

CONCLUSION

Olfactory dysfunction is common in patients with CRS. Age, nasal polyposis, smoking status, and asthma are significant predictors of olfactory dysfunction. Neither a history of prior endoscopic sinus surgery nor a history of allergic rhinitis is associated with olfactory dysfunction. Septal deviation and inferior turbinate hypertrophy are associated with normal olfactory function. These findings may help to illuminate areas for future research to better understand the underlying mechanisms of olfactory dysfunction in patients with CRS.

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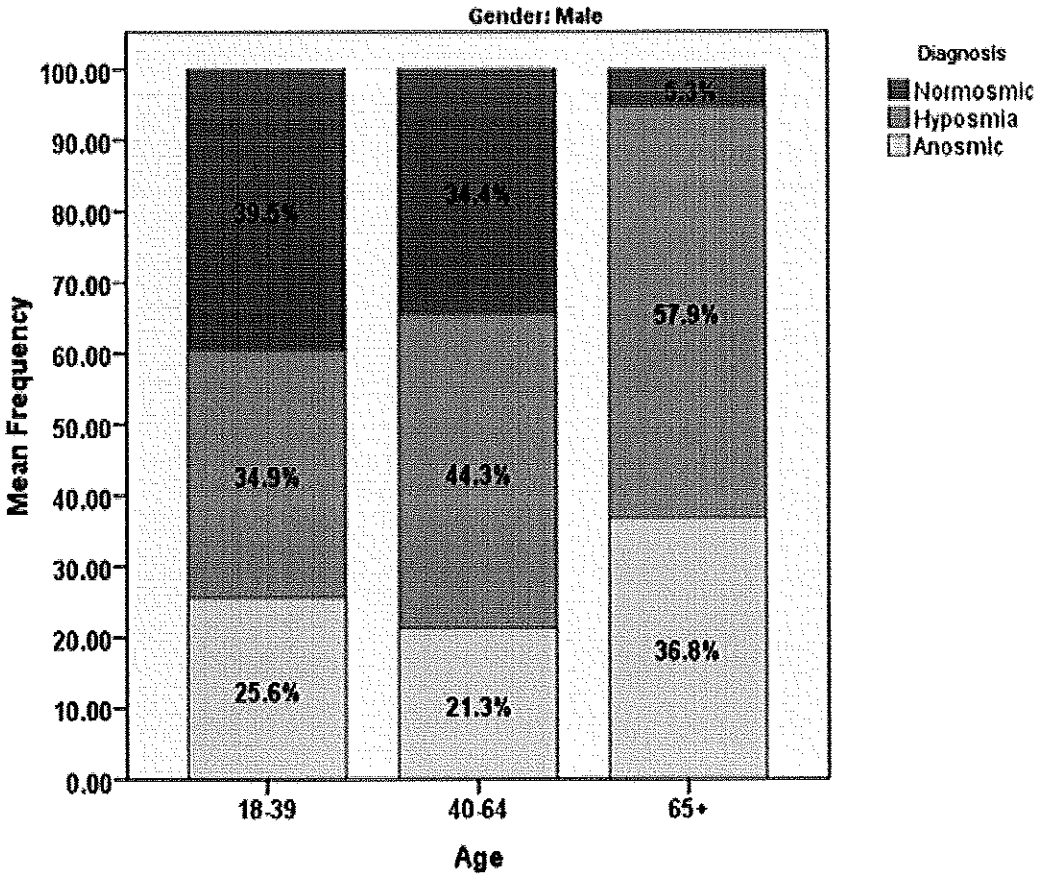

FIGURE I.

Prevalence of olfactory dysfunction for men across age categories: 18-39, 40-64, and 65+. The prevalence of hyposmia and anosmia in men with chronic rhinosinusitis was significantly higher in patients > 65 years old.

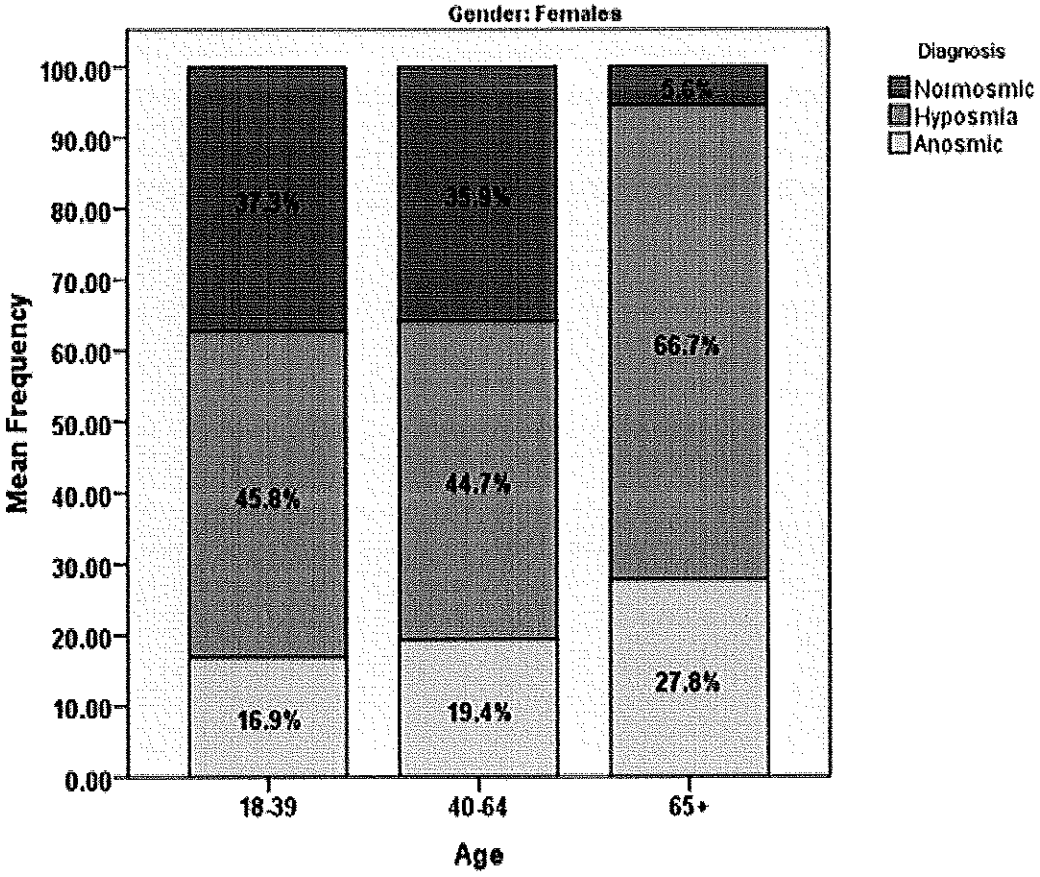

FIGURE II.
Prevalence of olfactory dysfunction for women across age categories: 18-39, 40-64, and 65+. The prevalence of hyposmia and anosmia in women with chronic rhinosinusitis was significantly higher in patients ≥ 65 years old.

Table I

Comparison of demographic and clinical characteristics of normosmic, hyposmic, and anosmic patients.

	Normosmic (N=122)	Hyposmic (N=166)	Anosmic (N=79)	P value
Age	43.8 +/- 12.5	49.8 +/- 13.6	49.4 +/- 13.8	**<0.001**
Gender				
Male	50% (61)	48.8% (81)	55.7% (44)	0.590
Female	50% (61)	51.2% (85)	44.3% (35)	
Clinical characteristics				
Nasal polyposis	18.0% (22)	33.7% (56)	74.7% (59)	**<0.001**
Asthma	29.5% (36)	33.1% (55)	69.6% (55)	**<0.001**
Aspirin Intolerance	4.1% (5)	7.2% (12)	24.1% (19)	**<0.001**
Allergic rhinitis	34.4% (42)	33.1% (55)	26.6% (21)	0.476
Septal deviation	31.1% (38)	28.3 (47)	7.6% (6)	**<0.001**
Turbinate hypertrophy	11.5% (14)	7.2% (14)	2.5% (2)	0.077
Prior sinus surgery	46.7% (57)	56.5% (94)	65.8% (52)	**0.026**
Smoker	5.7% (7)	8.4% (14)	7.6% (6)	0.684

Table II

Odds ratios, 95% confidence intervals (CI), and P values for each variable in the hyposmia and anosmia models.

Model	Variable	Odds Ratio (OR)	95% C.I.	P Value
Hyposmia	Age ≥ 65	10.03	2.30, 43.71	**0.002**
	Nasal polyposis	2.38	1.34, 4.23	**0.003**
Anosmia	Age ≥ 65	15.59	2.32, 104.86	**0.005**
	Nasal polyposis	13.21	5.68, 30.70	**<0.001**
	Current smoker	7.58	1.82, 31.55	**0.005**
	Asthma	4.21	1.81, 9.81	**0.001**
	Inferior turbinate hypertrophy	0.10	0.01, 0.91	**0.041**
	Septal deviation	0.19	0.06, 0.60	**0.005**

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Once-Daily Aminoglycoside Therapy: Potential Ototoxicity

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Current data indicate that once-daily aminoglycoside therapy is as efficacious as traditional multiple daily dosing and equally or less toxic. Our experience with once-daily gentamicin, 6 mg/kg of body weight, led to a 10% (3 of 33 patients) occurrence of documented ototoxicity after prolonged aminoglycoside exposure.

Many studies have been performed to define optimal serum aminoglycoside concentrations and dosing regimens and the causes of their associated nephro- and ototoxicity (1). However, dosing regimens leading to maximum efficacy and minimal toxicity have not been clearly delineated. Recently, there has been a plethora of literature reporting increased efficacy and less toxicity associated with less-frequent (once-daily) aminoglycoside dosing compared to multiple-daily-dosing regimens (5, 6, 9–11). Current available data indicate that once-daily aminoglycoside dosing is equal in efficacy to the multiple-dosing regimen and the occurrence of nephrotoxicity is equal to or lower than that with the multiple-dosing regimen. However, there are limited data regarding the impact of this dosing regimen on the development of ototoxicity. We present three patients who developed ototoxicity after receiving once-daily gentamicin at 6 mg/kg of body weight per day according to a protocol designed to achieve peak concentrations in serum between 15 and 24 mg/liter and trough concentrations less than 0.5 mg/liter.

Patient 1. Patient 1 was a 52-year-old, 60-kg female with a history of calcinosis cutis, Raynaud's phenomenon, esophageal motility disorder, sclerodactyly, and telangiectasia syndrome for 5 years. She had nonhealing ulcers on the dorsa of both feet and was admitted to the hospital for excision of the exposed metatarsal heads. A bone culture grew *Pseudomonas aeruginosa*. The patient was started on ticarcillin-clavulanic acid (3.1 g every 4 h) and gentamicin (360 mg every 24 h). With this patient's calculated creatinine clearance of 71 ml/min, a steady-state peak gentamicin level of 18 mg/liter and trough of 0.02 mg/liter were expected. The patient's reported initial peak and trough gentamicin concentrations were 16 and less than 0.5 mg/liter, respectively. The patient was discharged home on the above medications without any complaints. Three weeks later, she was readmitted with a diagnosis of diverticulitis. She continued to receive gentamicin, but the dose was reduced to 280 mg every 24 h because a subsequent peak gentamicin level was 27 mg/liter. On 280 mg every 24 h, the gentamicin peak concentration was 18 mg/liter. When she was discharged, the gentamicin was inadvertently discontinued for 9 days.

Gentamicin, 280 mg every 24 h, was then reinstituted. Three days later the patient complained of dizziness, light-headedness, and difficulty walking which she had been experiencing since discharge. Gentamicin was discontinued. An audiogram was normal, but the patient was unable to stand up in a dark room. An ear, nose, and throat physician felt she had oscillopsia indicating bilateral labyrinthine dysfunction. Further evaluation 6 weeks later demonstrated some difficulty with oculomotor tasks. There was no positional or spontaneous nystagmus. Cool-water irrigation produced very slight nystagmus. Ice water irrigation produced a weak response in both ears suggesting bilateral labyrinthine hypoactivity.

Patient 2. Patient 2 was a 71-year-old, 72-kg male admitted with a temperature of 103°F (ca. 39.4°C), rigors, difficulty urinating for 3 days, and right back pain radiating to his scrotum and right leg. Magnetic resonance imaging of the spine revealed discitis at L3-L4, and a bone scan was positive for vertebral osteomyelitis. Blood cultures from admission grew *Enterobacter aerogenes*, and the patient was placed on imipenem-cilastatin (500 mg every 6 h) and gentamicin (430 mg [6 mg/kg] every 24 h). He had a calculated creatinine clearance of 72 ml/min. Initial gentamicin peak, 12-h, and trough levels were 18, 1.1, and 0.5 mg/liter, respectively, which were close to an expected peak of 20 mg/liter and trough of 0.1 mg/liter. After 10 days of therapy, repeat peak and trough levels were 15 and 1.6 mg/liter, respectively. Because of an increase in serum creatinine (1 to 1.3 mg/dl), the dose was adjusted to 430 mg every 36 h for an additional 7 days. Because of an elevated peak gentamicin level of 32 mg/liter (trough = 1.1 mg/liter), the dose was further reduced to 400 mg every 36 h, which the patient received for an additional 11 days. The patient's serum creatinine returned to baseline.

After receiving 40 days of therapy, the patient complained of dizziness while ambulating, and during a physical therapy exam the patient was noted to have increased unsteadiness. The patient noted that he had been feeling dizzy while in the hospital. After discharge, he used a cane to walk most of the time. He described his unsteadiness as a sensation of being drunk and feeling as though the room were spinning. Magnetic resonance imaging of the brain and brain auditory evoked response were normal.

Electronystagmogram revealed mild left nystagmus in the right and left lateral positions. Ice water calorics showed markedly reduced responses in both ears consistent with ototoxicity. A computerized rocking chair test indicated a partially decompensated peripheral vestibular disorder with good central suppression. Audiometry revealed a bilateral moderate to severe sensorineural hearing deficit.

Patient 3. Patient 3 was an 80-year-old, 70-kg male who developed the sudden onset of midabdominal pain, nausea, fever, and chills. He was hypotensive on admission with a blood pressure of 80/50 mm Hg. Physical examination revealed mild epigastric tenderness. An endoscopic retrograde cholangiopancreatography revealed a stone blocking the bile duct, and a

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stent was placed. Blood cultures from admission grew *Escherichia coli*. Bile cultures grew *E. coli*, *P. aeruginosa*, *Enterococcus faecium*, and *Pseudomonas putida*. The patient was treated with imipenem-cilastatin (500 mg every 6 h) and gentamicin (420 mg [6 mg/kg] every 24 h) on the basis of a calculated creatinine clearance of 57 ml/min. While initial steady-state gentamicin peak and trough concentrations were expected to be 20 and 0.25 mg/liter, respectively, the actual peak was 15 mg/liter, the 12-h level was 3.3 mg/liter, and the trough was 0.7 mg/liter. Based upon pharmacokinetic calculations, these gentamicin levels indicate that the patient had an increased volume of distribution. However, the clearance was not different from that estimated. Following 12 days of therapy, the patient's estimated creatinine clearance decreased to 44 ml/min and subsequent gentamicin levels were a peak of 19 mg/liter and trough of 1.3 mg/liter. The patient received a total of 16 days of therapy. He was discharged from the hospital feeling well and had no vestibular complaints. After being off gentamicin for 2 weeks, he developed some dizziness and felt unsteady. An electronystagmogram revealed normal caloric responses in both ears but unilateral right-side weakness consistent with a peripheral vestibular neuropathy. Magnetic resonance imaging of the brain was normal. An audiogram revealed mild to moderate sensorineural hearing loss from 500 to 8,000 Hz (low and high frequencies) in the right ear along with poor speech discrimination; the left ear had a mild to moderate high-frequency hearing loss (3,000 to 8,000 Hz) and excellent speech discrimination.

Aminoglycoside antibiotics have been shown to produce ototoxicity which may be irreversible in both humans and experimental animals (2). Ototoxicity can take the form of auditory and/or vestibular changes resulting in destruction of sensory hair cells in the cochlea and vestibular labyrinth (2). The initial stages of auditory toxicity involve selective destruction of the outer hair cells of the organ of Corti. In this early stage of toxicity, the damage is usually limited to higher frequency levels (4,000 to 8,000 Hz) and does not affect frequencies utilized in conversational hearing (4). The toxic changes are generally reversible at this stage. If the insult is allowed to progress, the inner hair cells of the cochlear apex become damaged (12). Hearing impairment then occurs at lower frequencies, and conversational hearing is compromised. At this later stage, the deficit is generally permanent or only partially reversible. Vestibular toxicity generally parallels cochlear damage and is usually manifested by vertigo, nausea, dizziness, and nystagmus (12).

The precise mechanism of hair cell destruction in both forms of ototoxicity is unclear. The incidence of hearing loss ranges from 2 to 25% (7). This wide range may be due in part to the lack of baseline and subsequent auditory determinations and to the absence of a universally accepted standard for defining drug-induced ototoxicity. In addition, because the vast majority of patients receiving aminoglycoside therapy are lost to follow-up and the symptoms are ambiguous, the permanent or transient nature of such an adverse reaction is also not known. Several factors have been associated with a higher incidence of ototoxicity, including (i) duration of therapy (>8 days), (ii) cumulative dose, (iii) total daily dose, (iv) peak and trough serum drug concentrations, (v) concurrent diuretic therapy, (vi) underlying disease states, (vii) previous exposure to aminoglycoside therapy, (viii) increased age, and (ix) specific aminoglycosides (7, 12). However, recently it has been suggested that the accumulation of aminoglycosides in cochlear and vestibular tissues is related to prolonged exposure rather than to transient high concentrations in serum (3). Therefore, prolonged exposure of the hair cells to the aminoglycoside may account for the damage observed (3).

Studies evaluating once-daily aminoglycoside dosing have suggested that the incidence of ototoxicity is similar to or less than that with traditional dosing of aminoglycosides (3). However, many studies did not evaluate hearing, used various methods to establish ototoxicity, or used various doses (range, 3.8 to 7 mg/kg/day) or patients were on short courses of aminoglycoside therapy (3, 8). In one large prospective study evaluating over 2,000 patients receiving once-daily aminoglycoside dosing (7 mg/kg/day), the average length of therapy was only 4.5 days (8). Only 808 (37%) patients received 6 or more days of therapy. However, one patient developed residual ototoxicity after receiving 5 weeks of therapy. Patients were followed up clinically, and determination of ototoxicity was made on the basis of patient interviews and physical examinations.

All three patients that we report on received aggressive once-daily dosing of an aminoglycoside for a period of 2 weeks or greater (range, 16 to 40 days). Despite two patients' initial serum drug concentrations being within the guidelines for dosing once-daily aminoglycosides (defined by Nicolau et al. [8]), the total daily doses used in these three patients were higher than conventional dosing guidelines for patients with some degree of renal impairment. Because of the small number of patients, we cannot determine if the observed ototoxicity was due to the dose or prolonged exposure to the drug. We have rarely observed similar ototoxicity in patients receiving conventional dosing of aminoglycosides, even when prolonged therapy is administered. However, upon initiating once-daily aminoglycoside dosing in our institution, 3 of 33 patients (10%) developed aminoglycoside-induced ototoxicity within a 3-month period. The high incidence of this adverse effect may be related to the duration of aminoglycoside therapy, the aggressive high-dose regimen, or both. Consequently, we recommend that prudence be employed in using this new aggressive dosing regimen. In addition, dosage adjustments may be required, and practitioners should pay particular attention to the development of ototoxicity. This may be especially important in patients receiving high doses and prolonged courses of aminoglycoside therapy.

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STEROID CATARACT

BY

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Black, Oglesby, von Sallmann, and Bunim (1960) first described posterior subcapsular lens opacities in seventeen of 44 patients suffering from rheumatoid arthritis who were maintained on long-term corticosteroid therapy, and later (Oglesby, Black, von Sallmann, and Bunim, 1961) they expanded the series to include patients suffering from a variety of rheumatic disorders. Subsequently other workers have described single cases or series (Hart, Casey, and O'Riordan, 1961; Pfahl, Makley, McCoy, and Rothermich, 1961; Toogood, Dyson, Thompson, and Mularchyk, 1962) covering many of the disorders frequently treated with corticosteroid therapy. When Crews (1963) compared 79 patients treated with corticosteroid therapy, of whom 52 were suffering from classical rheumatoid arthritis, with 171 patients who had never received corticosteroid therapy, of whom 34 had rheumatoid arthritis, he found bilateral posterior subcapsular lens opacities in 25 of the steroid-treated group and five in the non-steroid group; he considered that those in the steroid group had features differentiating them from the control group.

None of these studies contained an appreciable number of patients suffering from juvenile rheumatoid arthritis (Still's disease), and it was therefore felt worthwhile to review a series of such patients maintained on corticosteroid therapy and to compare them with adult patients.

Materials and Methods

At this Unit a combined eye and joint clinic is held primarily for reviewing all patients with Still's disease and patients who are on antimalarial therapy to exclude eye complications, and also for easy referral of rheumatic patients who have ocular symptoms or signs. For the purpose of this study it was decided to re-examine all cases of Still's disease who had been on maintenance corticosteroid therapy for at least the preceding year. Their names were obtained from the special register which is kept for all patients who receive more than 3 months of corticosteroid therapy. There were 53 patients suffering from definite Still's disease (criteria of Ansell and Bywaters, 1959*) who had never had any evidence of iridocyclitis and four who had iridocyclitis affecting one eye only. An equivalent number of patients suffering from classical rheumatoid arthritis (A.R.A. criteria, 1959) who had also been on corticosteroid therapy for a minimum of one year were asked to attend the combined clinic; these were selected only by reason of living within easy access of the base hospital. A control series comprised 48 patients with classical rheumatoid arthritis who attended the combined clinic on account of anti-malarial therapy during the period of the study and had never received corticosteroid therapy.

On this occasion, as part of their routine clinical check, the patients were asked for eye symptoms and then examined with an ophthalmoscope (using a +12 dioptre lens) by the clinician, whose findings were recorded independently. The patients were then reviewed by the ophthalmologist of the team (W.K.S.); the pupils were dilated and examined with the slit-lamp microscope. Viewed with a suitable plus lens with the ophthalmoscope, even an early steroid cataract can usually be detected in a dark room without dilating the pupil and without specialized ophthalmic training; it will be seen as a dark spot perhaps 1 mm. or less in diameter in the centre of the pupil against the red reflex of the retina. By parallax the spot can be confirmed to lie in the posterior cortex. At this early stage the vision is little affected but later, as the opacity increases in size, detection becomes obvious and sight increasingly blurred. When examined with the slit-lamp microscope with the pupil dilated, the opacity can be recognized at a very early stage by retro-illumination. By focal illumination it is seen to lie subcapsularly

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* Criteria for diagnosis of definite Still's disease. Onset of polyarthritis before the age of 16 years involving four or more joints for a minimum of 3 months or, when less than four joints are involved, a biopsy of synovial membrane showing histology compatible with a diagnosis of rheumatoid arthritis in the absence of evidence of other related diseases, such as rheumatic fever, psoriasis, systemic lupus erythematosus, etc.

in the posterior cortex on or near the axial line as one or more discrete grey dots with or without adjacent vacuoles. Polychromatic lustre may be present or absent. Later the dots coalesce into a plaque which gradually spreads peripherally and axially. The cataract in one of our cases, however, followed a different and unusual pattern.

Case 7, a boy who came under our care and commenced systemic corticosteroid therapy in 1961 at the age of 4 years, had severe Still's disease of 3 years' duration. At this time the right eye was entirely normal and the vision 6/6. The left eye was blind because of old iridocyclitis, band keratopathy, and complicated cataract—the typical triad of Still's disease (Smiley, May, and Bywaters, 1957). In early December, 1964, the patient noticed some fleeting fluctuation in the vision of the right eye but nothing abnormal was found on examination. At this stage he had been on systemic prednisone for 3¾ years, and although he had received 20 mg. daily for 14 days initially he was rapidly reduced to 5 mg. daily and later to 4 mg. daily, the mean daily dosage being 4·7, and the total amount given up to December, 1964, being 6·2 g. He went home on holiday for 10 days at Christmas and during this time his sight dramatically deteriorated. When he returned to hospital early in January, 1965, his sight was a blurred 6/60 with a myopic correction and the right lens was cataractous. Unlike the usual early steroid cataract, there was a diffuse haze throughout the lens cortex composed of tiny punctate opacities, with the suggestion of a denser plaque at the posterior pole. The retina could be seen only vaguely through the lens haze. The eye was free from any evidence of inflammation. During the next few months the generalized lens haze became less dense and more typical steroid opacities developed subcapsularly at the posterior and anterior poles. For some time before and since the development of the cataract the arthritis had been more active, the plasma proteins have shown a progressive fall in serum albumin, proteinuria has developed, and he has become nephrotic as a result of amyloidosis. How far his general deterioration predisposed to the unusual steroid cataract it is impossible to say.

Before a diagnosis of steroid cataract can be made, other causes of posterior subcapsular opacities have to be considered, *e.g.* familial, traumatic, complicated, toxic, radiational, and senile. It is only in cases in which these factors have been excluded and which have the characteristic appearance (Crews, 1963) that a firm diagnosis can be established.

Results

52 of the 53 children without iridocylitis together with the four cases of Still's disease with unilateral iridocyclitis and 57 cases of classical rheumatoid arthritis were examined during this study. Among these 113 patients on corticosteroid therapy, thirteen were found to have early lens opacities, all of which were subsequently confirmed as typical posterior subcapsular steroid cataracts. No cataract was found that had not been suspected by the rheumatologist. The cataracts were present in similar numbers among the patients with Still's disease and those with rheumatoid arthritis, giving an overall incidence of 11 per cent. (Table I). No similar cataract was found in the 48 patients with classical or definite rheumatoid arthritis who were on antimalarial therapy and had never received corticosteroid therapy.

TABLE I
INCIDENCE OF POSTERIOR SUBCAPSULAR CATARACT IN 113 PATIENTS ON LONG-TERM CORTICOSTEROID THERAPY

Diagnosis	Posterior Subcapsular Cataract		Total No. of Cases	Percentage with Posterior Subcapsular Cataract
	Present	Absent		
Total	13	100	113	11
Still's Disease	7	49	56	12·5
Rheumatoid Arthritis	6	51	57	10·5

With the exception of Case 7 in whom there was only one eye at risk because of chronic iridocyclitis in the other, all patients had bilaterial cataracts. As patients suspected of having steroid cataracts had been checked at the combined clinic before this study was commenced, the duration of therapy before the first sign of cataract appeared had already been established in some of them. In only one case was there evidence of a very marked interval between the involvement of the two eyes. This was Case 2, a girl suffering from Still's disease, who had been noted to have a cataract in one eye at the age of 6 after only 1¼ years of therapy, the second eye not becoming involved until 2½ years later. This is the earliest instance in which cataract was detected in our series. The other cases among the children occurred at intervals varying up to 5¾ years from the onset of therapy. The mean daily dose given to these patients, calculated as mg. prednisone, had varied between 4·7 and 20·2 mg. and the total amount given before the development of cataract varied between 2·9 and 40·6 g. (Table II).

TABLE II
DOSAGE AND DURATION OF CORTICOSTEROID THERAPY IN SEVEN PATIENTS WITH STILL'S DISEASE WHO DEVELOPED CATARACT

Case No.	Steroid Dosage (Prednisone)		Duration of Steroid Therapy (yrs)
	Total (g.)	Daily Average (mg.)	
1	12·8	9·8	3¼
2 (left eye)	2·9	6	1¼
2 (both eyes)	7·5	6	3¼
3	12·1	4·9	5¾
4	24·7	13·2	5
5	40·6	20·2	5¼
6	10·9	8·9	3¼
7	6·2	4·7	3¼

In adults the first cataract was not detected until 2¾ years after the institution of corticosteroid therapy and in the others after from 3 to 7 years of treatment: here the mean daily dose varied between 7·5 and 14·9 mg. daily, and total amount before the development of cataract varied from 8·1 to 29·7 g. prednisone or equivalent (Table III).

TABLE III
DOSAGE AND DURATION OF CORTICOSTEROID THERAPY IN SIX PATIENTS WITH RHEUMATOID ARTHRITIS WHO DEVELOPED CATARACT

Case No.	Steroid Dosage (Prednisone)		Duration of Steroid Therapy (yrs)
	Total (g.)	Daily Average (mg.)	
8	29·7	14·9	5½
9	19·0	11·4	4½
10	25·3	9·6	7
11	12·0	7·7	3½
12	8·1	7·5	2¾
13	16·1	13·8	4

For simplicity's sake all the corticosteroid therapy has been calculated in terms of mg. prednisone, although some patients had received intermittent therapy with other corticosteroid drugs, such as cortisone, hydrocortisone, methylprednisolone, triamcinilone, dexamethazone, and betamethazone. With the relatively small numbers available, there was no evidence that different corticosteroid preparations had different effects on cataract formation.

It proved possible to calculate the total and average daily dose of corticosteroid from the case records and treatment sheets of all patients who developed this complication and in 48 others. The duration of therapy was not significantly different in the two groups, but the average daily dose of prednisone was somewhat higher, 11·5 mg. in those who developed cataract compared with 8·8 mg. in those who did not (Table IV). Separating the children from the adults, the difference in dosage between those who developed cataract and those who did not becomes a little more striking among the juveniles where the overall dosage is lower (Table V). However, despite the suggestion that

TABLE IV
PRESENCE OF CATARACT RELATED TO DURATION AND AMOUNT OF THERAPY

Posterior Subcapsular Cataract	Patients on Corticosteroid Therapy	Average Duration of Therapy (yrs)	Average Daily Dosage of Prednisone (mg.)
Present	13	4½	11·5
Absent	48	5	8·8

TABLE V
DIAGNOSIS AND PRESENCE OF CATARACT RELATED TO DURATION AND AMOUNT OF THERAPY

Diagnosis	Posterior Subcapsular Cataract	No. of Cases	Average Duration of Therapy (yrs)	Average Daily Dosage (mg.)
Still's Disease	Present	7	4	10·6
	Absent	15	7	7·7
Rheumatoid Arthritis	Present	6	4½	12·2
	Absent	33	4¾	9·5

dosage is important, eight of the thirteen affected patients had never received more than 10 mg. daily, so it is unwise to ignore the possibility of this complication in patients on low dosage schedules (Table VI).

TABLE VI
AVERAGE DOSAGE AND DURATION OF CORTICOSTEROID THERAPY IN THIRTEEN PATIENTS WITH POSTERIOR SUBCAPSULAR LENS OPACITIES

Duration of Therapy (yrs)	Average Daily Dosage (mg.)		
	Less than 10	10-15	More than 15
Under 1			
1 to 4	6	2	
5 and More	2	2	1

Only two patients complained spontaneously of any visual disturbance, although on direct questioning, seven of the thirteen had mild disturbances of vision, usually transient slight blurring or impaired long-distance vision. During the course of the year, vision has become increasingly impaired in the two patients who complained of symptoms.

Discussion

The incidence of cataract in the general population is extremely low, so that a report on a hospital in-patient inquiry for the year 1958 (Sorsby, 1962) shows an incidence of one cataract per 10,000 of the general population in the 0 to 15-year age group. The presence, therefore, of cataract in seven of 56 juveniles is very much greater than would be expected. It has been suggested that this might be related to the underlying disease process rather than to therapy. However, over the last 16 years, over 300 children have been examined and cataract has been found only in those with chronic iridocyclitis or on corticosteroid therapy. In the control group of adults not receiving corticosteroid therapy, no cataracts were found, although the rheumatoid disease was classical and of moderate severity.

The incidence of 11 per cent. in this series is low compared with that cited in the literature (Black and others, 1960; Oglesby and others, 1961). This may well be explained by the fact that this was an unselected group of patients and also by our policy of keeping corticosteroid dosage to a minimum level: it is extremely rare for patients here to receive 15 mg. or more of prednisone daily, the majority being maintained below 10 mg. daily. There does not seem to be any overall difference in cataract formation between the juvenile and the adult group, but it was found that children may develop this complication at a lower dose and in a shorter time. Thus, among the children, one patient on 6 mg. daily developed a cataract after 1¼ years and a second one did likewise on 4·9 mg. daily after 5¾ years, whereas in the adult group the shortest duration of time was 2¾ years and the lowest dose 7·5 mg. daily. However, taking the group as a whole, the difference between the average daily dose and duration of corticosteroid therapy between the patients with subcapsular cataract was not significant.

Once the clinician became used to looking for this complication it was found relatively easy to diagnose it at routine examination. As our observation of the patients suggests that cataract can develop quite quickly, *i.e.* in 2 to 3 months, it would seem that examination of the lens should form part of the routine 3-monthly check for patients on corticosteroid therapy even when they are maintained at a level below 10 mg. daily.

It is not sufficient to wait until patients complain of visual disturbance, as by this time the cataract is well developed. Our aim is to reduce the dose of corticosteroid as soon as any sign of lens change occurs and in a number in whom this has been done there has been no increase in the size of the opacities or deterioration in vision over 1 year.

Although only two patients in this unselected group experienced deterioration of vision, a number of patients receiving maintenance corticosteroid therapy for rheumatoid arthritis, and other chronic conditions such as asthma, have also been seen with this complication. In several of these cases removal of the steroid cataract (W.K.S.) has been required to restore vision when blindness threatened.

Summary

(1) A total of 113 patients, 56 children with Still's disease and 57 adults with classical rheumatoid arthritis, receiving maintenance corticosteroid therapy, was examined, together with 48 patients with classical rheumatoid arthritis not on corticosteroids. Thirteen patients, seven with Still's disease and six with adult rheumatoid arthritis, were found to have posterior subcapsular lens opacities. These were all receiving corticosteroid therapy.

(2) The duration of therapy seemed to be less important than the average daily dose; while this tended to be higher in the patients developing cataract, particularly among the juveniles, eight of the thirteen cataracts occurred in patients whose daily dose of prednisone was 10 mg. or less.

(3) In the hope that cessation or reduction of steroid administration may inhibit or slow down the progress of an early cataract, this condition must be looked for routinely and repeatedly in patients on corticosteroids. If there is any suggestion of an opacity, the patients should be referred to an ophthalmologist.

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La cataracte stéroïde

Résumé

(1) On a examiné 56 enfants atteints de maladie de Still et 57 adultes souffrant d'arthrite rhumatismale classique, en tout 113 malades soumis au régime corticostéroïde d'entretien ainsi que 48 autres malades atteints d'arthrite rhumatismale classique mais ne recevant pas de corticostéroïdes. On a trouvé que 13 malades, dont sept atteints de maladie de Still et six adultes atteints d'arthrite rhumatismale présentaient des opacités sous-capsulaires postérieures du cristallin. Tous ces malades étaient au régime corticostéroïde.

(2) La durée de la thérapie semblait être moins importante que la dose moyenne par jour; bien que celle-ci tendait à être plus forte chez les malades qui développaient une cataracte, surtout parmi les jeunes, huit sur les treize cataractes se sont produites chez des

malades qui recevaient 10 mg. de prednisolone ou moins par jour.

(3) En espérant que l'interruption ou la réduction de la thérapie stéroïde puisse inhiber ou ralentir la formation d'une cataracte naissante, il faut rechercher cette condition régulièrement et à maintes reprises et, au moindre soupçon, consulter un ophtalmologiste.

La catarata esteroide

SUMARIO

(1) Se examinaron 56 niños con enfermedad de Still y 57 adultos con artritis reumatoide clásica, un total de 113 enfermos, sometidos a la terapia corticosteroide de sostén, así como 48 enfermos con artritis reumatoide clásica que no recibían corticosteroides. En trece enfermos—siete con enfermedad de Still y seis adultos con artritis reumatoide—se encontraron opacidades subcapsulares posteriores del cristalino. Todos estos enfermos recibían corticosteroides.

(2) La duración del tratamiento pareció tener menor importancia que la dosis diaria media: aunque ésta tendió ser más fuerte en enfermos que desarrollaron catarata, particularmente en los jóvenes, ocho de las trece cataratas se produjeron en enfermos recibiendo 10 mg. o menos de prednisolona al día.

(3) En la espera de que la interrupción o la reducción de la terapia esteroide pueda inhibir o retardar la formación de una catarata naciente, se debe buscar esta condición regular y repetidamente y, a sospechar una opacidad, consultar el oculista.

Curriculum Vitae

Ernest P. Chiodo, M.D., J.D., M.P.H., M.S., M.B.A., C.I.H.

Diplomate of the American Board of Internal Medicine
Diplomate of the American Board of Preventive Medicine in Occupational Medicine
Diplomate of the American Board of Preventive Medicine in Public Health and General Preventive Medicine
Diplomate of the American Board of Industrial Hygiene as a Certified Industrial Hygienist
Graduate Biomedical Engineer
Graduate Toxicologist

PROFESSIONAL ADDRESSES:

Detroit Area:	Chicago:	Palm Beach, Florida:
Ernest Chiodo P.C. 35770 Harper Avenue Clinton Township, Michigan 48035 Tel: (586) 746-1761	Ernest Chiodo P.C. 221 N. LaSalle Street Suite 854 Chicago, IL 60601 Tel. (312) 351-0717	701 S. Olive Suite 113 W. Palm Beach, FL 33401 Tel. (561) 603-7701

Email: epchiodo@gmail.com
www.ernestpchiodo.com

EDUCATION:

University of Chicago
Master of Business Administration
With a Concentration in Economics — December 9, 2011

Wayne State University
Master of Science in Occupational and Environmental Health Sciences
With Specialization in Industrial Toxicology — December 23, 2009

University of Chicago
Master of Science in Threat Response Management
(Scientific tract in biological, chemical, and radiological defense) — June 12, 2009

Wayne State University Department of Biomedical Engineering
College of Engineering and School of Medicine
Master of Science in Biomedical Engineering — December 20, 2007

Harvard University School of Public Health
Master of Public Health — June 8, 1989

Wayne State University Law School
Juris Doctor — June 14, 1986

Wayne State University School of Medicine
Doctor of Medicine June 8, 1983

Kalamazoo College
Bachelor of Arts June 14, 1980

SPECIALTY TRAINING:

Mini Residency in German Social Medicine at the
Landesversicherungsanstalt Baden in Karlsruhe, Germany May 1992

Resident Physician in Internal Medicine
Providence Hospital
Southfield, Michigan July 1, 1989 to June 30, 1992

Resident Physician in Diagnostic Radiology
Detroit Medical Center
Detroit, Michigan July 1, 1983 to June 30, 1985

PROFESSIONAL ACTIVITY:

Ernest Chiodo P.C.
Professional corporation licensed to practice medicine and law
in the State of Michigan. 1989-Present

Medical professional activities through Ernest Chiodo P.C. have included acting as the Chief Medical Director of the Visiting Nurse Association of Southeastern Michigan; Medical Director of the City of Lansing Police and Fire Pension Board; Medical Director of the City of Lansing General Employee Retirement System; Medical Director of the Fire and Police Pension System of the Charter Township of Clinton, Michigan; International Advisor on Bird Flu to Goodrich Corporation (Formerly B.F. Goodrich); the clinical practice of internal medicine; the clinical practice of preventive medicine; the practice of occupational and environmental medicine; the practice of occupational, environmental, and industrial toxicology; the practice of industrial hygiene; biomedical engineering; and biostatistics and epidemiology. Legal professional activities have included the practice of toxic tort law as well as the practice of health care law in the representation of physicians, nurses, and other health care professionals. Areas of representation have included licensure and peer review issues; contract review and negotiation; Stark Amendments; Sherman and Clayton Anti-trust Acts; new medical technology assessment; and co-counsel activities in medical malpractice defense. Health care law activities have included consultation and general counsel activities for health care entities including service as legal counsel to the Medical Staff and Internal Medicine Department of Henry Ford Bi-County

Hospital. Biomedical engineering activities include determination of causation of disease in relation to claimed physical forces and energy. Health care economics activity has included analysis of reasonableness of charges for medical devices and services.

Center for Health Outcomes and Evaluations
Michigan Peer Review Organization
Clinical Coordinator 1995-1996

The activities for the Center for Health Outcomes and Evaluations, a division of the Michigan Peer Review Organization, involved conducting clinical outcomes research. This research was contracted for by the Health Care Financing Administration. The research compared clinical outcomes in hospitals throughout the State of Michigan. The information gained through the research was also presented to collaborating hospitals and their medical staffs in an effort to increase quality of care for Medicare beneficiaries.

Medical Director
Detroit Health Department 1993-1995

Service as the Medical Director and Manager of the 1,200 person Detroit Health Department and advisor to the Mayor of the City of Detroit on health care and public health issues.

Medical Director
House Physician Program
Bi-County Community Hospital 1992-1993

Medical Director of the physicians servicing the medical needs of patients admitted to the non-teaching service of Bi-County Community Hospital.

Medical Director and Chief Executive Officer
General Health Corporation 1985-1989

Home health care service caring for serious traumatically brain injured and quadriplegic patients.

PROFESSIONAL LICENSURE:

Physician and Surgeon
Michigan, Illinois, Florida and New York

Attorney and Counselor
Michigan and Illinois

TRADE LICENSURE:

Residential Builder
Michigan

SPECIALTY BOARD CERTIFICATION:

Diplomate of the American Board of Internal Medicine
(First recertification May 2000)
(Second recertification November 2012)

Diplomate of the American Board of Preventive Medicine in Occupational Medicine

Diplomate of the American Board of Preventive Medicine in Public Health and General Preventive Medicine

Diplomate of the American Board of Industrial Hygiene as a Certified Industrial Hygienist

MEDIATION CERTIFICATION:

Completion of requirements of Michigan Court Rule 2.411 to serve as a court appointed mediator in Michigan.

MEDICAL REVIEW OFFICER CERTIFICATION:

Medical Review Officer Certification through the Medical Review Officer Certification Council.
Date of Original Certification: February 21, 2006
Recertification: October 7, 2011

RATINGS:

BV Rating by Martindale-Hubbell Ratings[SM]

"A BV Rating is an indication of an exemplary reputation and well-established practice. A typical lawyer is in mid-career, with a significant client base and high professional standing."

"CV, BV and AV are registered certification marks of Reed Elsevier Properties Inc., used in accordance with the Martindale-Hubble certification procedures, standards and policies."

PROFESSORSHIPS AND FACULTY APPOINTMENTS:

Assistant Clinical Professor of Internal Medicine, Family Medicine and Public Health
Wayne State University School of Medicine
Detroit, Michigan
1994-2013

Adjunction Assistant Professor of Industrial Hygiene and Industrial Toxicology
Eugene Applebaum College of Pharmacy and Health Science
Department of Occupational and Environmental Health Sciences
Wayne State University
Detroit, Michigan
2009-2013

Adjunct Professor of Law
John Marshall Law School
Chicago, Illinois

Adjunct Professor of Law
Loyola University of Chicago Law School
Chicago, Illinois

FELLOWSHIPS:

Royal Society of Medicine
Overseas Fellow
Elected 20000

PROFESSIONAL ORGANIZATIONS:

State Bar of Michigan
Environmental Law Section of the State Bar of Michigan
Royal Society of Medicine (U.K.)
American Industrial Hygiene Association
Chicago Bar Association
University of Chicago Booth Health Care and Biopharma Round Table. Co-Chariperson.
Florida Medical Association
Full Member Society of Toxicology

PROFESSIONAL ORGANIZATION LEADERSHIP:

Vice Chairman, By-laws Committee: Wayne County Medical Society 1994-1995
Chairman, By-laws Committee: Harper Hospital 1995-1997
Member, Public Health Committee: Wayne County Medical Society 1993-1996
Member, Medical-Legal Committee: Wayne County Medical Society 1994-1996
Member, Michigan Department of Public Health Liaison Committee: Michigan State Medical Society 1994-1996
Chief Medical Director: Visiting Nurse Association of Southeastern Michigan 1994-1999, 2002-2005.
Medical Director Emeritus: Visiting Nurse Association of Southeastern Michigan 2005-Present
Member, By-laws Committee: Henry Ford Bi-County Hospital 2004 -2010
Member, Infection Control Committee: Henry Ford Bi-County Hospital 2003-2010
Vice Chairman and Chairman of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan. 2006-2012.
Council Member of the Environmental Law Section of the State Bar of Michigan (The Council is the governing body responsible for general supervision and control of the affairs of the Section). 2006 –2012
Board of Directors of the Michigan Industrial Hygiene Society 2006-2007.
President-Elect of the Michigan Industrial Hygiene Society 2007.
President of the Michigan Industrial Hygiene Society 2008.
Co-Chairman of the University of Chicago Booth Health Care and Biopharma Round Table.
Member of Board of Directors of the University of Chicago Booth Alumni Club

COMMUNITY SERVICE ORGANIZATION LEADERSHIP:

Michigan Cancer Foundation Board of Trustees (Term 1994-1997)
Tri-cities Tobacco Action Coalition Advisory Board 1994-1995
Tobacco Free Michigan Active Doctors Advisory Committee 1994-1995
Wayne County American Cancer Society Board of Directors 1994-1996

SOCIAL AND ALUMNI ORGANIZATIONS:

Harvard Club of Eastern Michigan
Union League Club of Chicago
University Club of Chicago
Harvard Club of New York City
University of Chicago Booth alumni Club

RESEARCH:

Stitch Entrapment of Swan-Ganz Catheter during Cardiac Surgery. Presented at the American College of Physicians Associates Meeting, May 11, 1990 and the Michigan Chapter Meeting, Traverse City, Michigan, October 11-14, 1990.

Altered Cerebral Dominance in an Atopic Population. Presented at the American College of Physicians Associates Meeting, May 11, 1990 and orally presented at the Michigan Chapter Meeting, Traverse City, Michigan, October 11-14, 1990.

Code Status Determination: An Analysis of Decisions by Health Care Professionals. Orally Presented at the American College of Physicians Associates Meeting, May 10, 1991.

A Case of Ovarian Carcinoma with Concomitant Weakness and Dysphagia and Biopsy Proven Myositis. Presented at the American College of Physicians Associates Meeting, May 10, 1991 and the Michigan Chapter Meeting, Traverse City, Michigan. September 26-28, 1991.

A Case of Persistent Hyponatremia in a Patient with a Mediastinal Mass on CT. Presented at the American College of Physicians Associates Meeting, May 10, 1991 and the Michigan Chapter Meeting, Traverse City, Michigan, September 26-28, 1991.

Refractory Pneumocystis Carini Pneumonia in a HIV Positive Patient Successfully Treated with 566c80 a 1,4 Hydroxynapthoquinone with a Broad Spectrum Antiprotozoal Activity. Presented at the American College of Physicians Associates Meeting, Dearborn, Michigan, May 1992.

PRESENTATIONS AND LECTURES:

Health Care Regulations in Present Day Medical Practice. Presented as part of the Current Problems in Medicine 1990 course sponsored by St. John Hospital and Medical Center. Hyatt Regency, Dearborn, Michigan on October 21, 1990. Approved for 4.5 hours category 1-CME credit.

The Medical, Legal, and Financial Consequences for Catastrophic Injury. Presented at the Eastern Michigan Area Social Security Manager's Meeting at the request of the Department of Health & Human Services. Pontiac, Michigan on June 11, 1990.

Advisor and judge of the 1st Annual Wayne State University School of Medicine - Wayne State University Law School Mock Medical Malpractice Trial . Sponsored by the American Medical Association and approved for law school credit. Gordon Scott Hall, Wayne State University School of Medicine, February 27, 1991.

Advisor and judge of the 2nd Annual Wayne State University School of Medicine - Wayne State University Law School Mock Medical Malpractice Trial). February 25, 1992.

Krankenversicherung in die Vereinigten Staaten (Health Insurance in the United States). Lecture given in German to the physicians of the medical division of the Landesversicherungsanstalt Baden in Karlesruhe, Germany, May 12, 1992. The Landesversicherungsanstalt is the German equivalent of the Social Security Administration.

Privatization of Government. Panel discussion given at the Michigan Association of Counties meeting at the Grand Hotel on Mackinaw Island, Michigan on August 20, 1993. My portion of the discussion focused on the arguments for and against privatization of public health services at the county and municipal level.

Law for the Industrial Hygienist. Lecture given in the Department of Occupational and Environmental Health in the School of Allied Health and Pharmacy, Wayne State University on November 30, 1993.

Teen Pregnancy, Sex, and Health Care. Workshop presented at the Youth Anti-Crime Summit: A Blueprint for Our Future. Wayne County Community College in Detroit, Michigan on October 23, 1993.

The Obstetrician and Gynecologist as a Primary Care Physician. Grand Rounds presented to the staff of Hutzel Hospital and the Department of Obstetrics and Gynecology of Wayne State University School of Medicine. February 8, 1994 at Hutzel Hospital in Detroit, Michigan.

Breast and Cervical Screening: What the Pharmacist Needs to Know. Lecture presented at the 1994 interim meeting of the Michigan Pharmacists Association at the Hyatt Regency in Dearborn, Michigan on February 18, 1994.

Career Opportunities in Health Care Law. Panel discussion with Edward B. Goldman, Medical Center Attorney for the University of Michigan, and Bettye S. Elkins, health care attorney with the firm of Dykema Gossett, presented to the Health Care Law Society of the University of Michigan Law School. March 9, 1994 in Hutchins Hall, University of Michigan Law School.

Community Health Grand Rounds. Member of panel discussion presented at the Detroit Health Department. Sponsored by the Southeastern Michigan Health Association and the Resource for Public Health Policy of the University of Michigan School of Public Health. March 10, 1994.

Environmental Toxicology. General Internal Medicine Ambulatory Grand Rounds. Wayne State University School of Medicine. March 18, 1994 in the Morse Auditorium of Harper Hospital.

Introduction to Occupational and Environmental Medicine. Internal Medicine Grand Rounds. March 22, 1994. St. John Hospital and Medical Center. Detroit, Michigan.

What the Family Practitioner Should Know About the National Practitioner Data Bank. Presented to the Department of Family Practice. April 1, 1994. St. John Hospital and Medical Center. Detroit, Michigan.

Public Health Case Studies. Presented to the University of Michigan Family Practice Residency Program. April 6, 1994. University of Michigan Chelsea Family Practice Center. Chelsea, Michigan.

Teen Pregnancy: A fresh approach to an old problem. Sponsored by the Wayne State University School of Medicine, the Wayne County Medical Society, and the March of Dimes Birth Defects Foundation, Southeastern Michigan Chapter. Approved for 3.5 continuing medical education credit hours in category 1. Presentation of the public health considerations in the session entitled Contraception versus Abstinence. April 27, 1994. Wayne County Medical Society Building. Detroit, Michigan.

The Future of the American Health Care System. Presented to the German-American Business Network. Sponsored by the German Consulate in Detroit. May 5, 1994. Hyatt Regency. Dearborn, Michigan.

The Future of Health Care in Detroit. Presented at the Henry Ford Health System Planning and Strategic Development Division Retreat. May 9, 1994. 1 Ford Place. Detroit, Michigan.

Was Napoleon Murdered by Arsenic Poisoning? Presented to the Napoleonic Society of America. September 16, 1994. Union League Club. Chicago, Illinois.

Is the World Safe for Children?: Environmental hazards and Their Impact. Presented at the Third Annual Medstart Conference. University of Michigan Medical School. January 21, 1995. Townsley Center. Ann Arbor, Michigan.

Treatment and Management of Arthritis in the Workplace. Presented at the Wayne County Medical Society conference on Arthritis in the Workplace. Co-sponsored for CME credits by the Medical and Public Health Issues Committee of the Wayne County Medical Society and the American College of Occupational and Environmental Medicine. January 28, 1995. Wayne County Medical Society Building. Detroit, Michigan.

Law and Medicine: What they didn't teach you in medical school. Presented for ambulatory grand rounds at the Veteran's Administration Medical Center in Allen Park, Michigan. April 25, 1995. Also presented to the following groups: Henry Ford Hospital Department of Urology. Henry Ford Hospital in Detroit, Michigan. June 14, 1995. Pediatric Grand Rounds. Children's Hospital of Michigan in Detroit, Michigan. August 25, 1995. Department of Physical Medicine and Rehabilitation. William Beaumont Hospital in Royal Oak, Michigan. September 15, 1995. Internal Medicine Grand Rounds. Grace Hospital in Detroit, Michigan. September 21, 1995. Wayne State University School of Medicine Department of Otorhinolaryngology. Harper Hospital in Detroit, Michigan. September 23, 1995. University of Michigan School of Medicine Department of Physical Medicine and Rehabilitation. University of Michigan Hospital in Ann Arbor, Michigan. September 28, 1995. Wayne State University School of Medicine Department of Neurology. Harper Hospital in Detroit, Michigan. October 6, 1995. Department of Obstetrics and Gynecology Grand Rounds. St. Joseph Mercy Hospital. Ann Arbor, Michigan. October 19,

1995. Department of Obstetrics and Gynecology Grand Rounds. University of Michigan Hospital. Ann Arbor, Michigan. Department of Ophthalmology. University of Michigan School of Medicine. Ann Arbor, Michigan. December 20, 1995. Department of Ophthalmology Grand Rounds, Henry Ford Hospital in Detroit, Michigan. February 16, 1996. Department of Surgery Grand Rounds. St. Joseph Mercy Hospital. Ann Arbor, Michigan. February 27, 1996. Michigan Association of Physicians from India. Dearborn Inn. Dearborn, Michigan. March 20, 1996. Wayne State University School of Medicine Department of Surgery Grand Rounds, Harper Hospital. Detroit, Michigan. May 4, 1996. Wayne State University School of Medicine Department of Internal Medicine. Grace Hospital. Detroit, Michigan. April 4, 1997. Wayne State University School of Medicine. Gordon Scott Hall. Detroit, Michigan. April 15, 1997. Michigan Psychiatric Society. Sheraton Inn. Ann Arbor, Michigan. April 24, 1997. Calhoun County Medical Society. Cedar Crest Banquet Center. Marshall, Michigan . February 13, 2001. Department of Family Practice Division of Occupational Medicine. Wayne State University School of Medicine. Detroit, Michigan. February 22, 2001. Department of Family Practice Division of Occupational Medicine. Wayne State University School of Medicine. Detroit, Michigan. August 15, 2001 St. John Hospital and Medical Center Department of Family Practice. St. Clair Shores, Michigan. October 23, 2001. Bon Secours Cottage Health Services. Henry Ford Health System. Grosse Pointe, Michigan. November 13, 2001. Department of Family Practice Division of Occupational Medicine. Wayne State University School of Medicine. Detroit, Michigan. November 14, 2001. Department of Psychiatry Grand Rounds. Henry Ford Health System. One Ford Place. Detroit, Michigan. November 15, 2001. Department of Pediatrics Grand Rounds. Henry Ford Health System. Henry Ford Hospital. Detroit, Michigan. December 13, 2001. Department of Surgery. Providence Hospital. Southfield, Michigan. February 14, 2002. Department of Family Practice Division of Occupational Medicine. Wayne State University School of Medicine. Detroit, Michigan. February 18, 2002. Pediatrics Residents. Henry Ford Health System. Henry Ford Hospital. Detroit, Michigan. May 3, 2002. Psychiatry Residents. Henry Ford Health System. One Ford Place. Detroit, Michigan. November 21, 2002. Psychiatry Residents. Henry Ford Health System. One Ford Place. Detroit, Michigan. January 15, 2004. Henry Ford Hospital Department of Cardiology. Detroit, Michigan. December 6, 2004. Harper Hospital Department of Cardiology. Detroit, Michigan. December 20, 2004. Southeastern Michigan Cardiology Fellows Club. Shula's Restaurant. Marriott Hotel. Troy, Michigan. November 16, 2005. Wayne State University School of Medicine. Gordon Scott Hall. Detroit, Michigan. January 24, 2006. Department of Dermatology, Wayne State University School of Medicine. VA Medical Center, Detroit, Michigan. January 17, 2007. Cardiology Fellows. William Beaumont Hospital. Royal Oak, Michigan. February 6, 2007. Residents and Medical Students. Henry Ford Bi-County Hospital. April 30, 2007. Medical Staff of William Beaumont Hospital Grosse Pointe. Grosse Pointe, Michigan. January 17, 2008. Department of Occupational and Environmental Medicine. University of Illinois at Chicago. Chicago, Illinois. May 7, 2008. Cardiology Fellows. William Beaumont Hospital. Royal Oak, Michigan. May 15, 2008. Cardiology Fellows. William Beaumont Hospital. Royal Oak, Michigan. June 2, 2009.

Euthanasia: Lessons from the past. Department of Internal Medicine Ambulatory Grand Rounds. Wayne State University School of Medicine. Harper Hospital. Detroit, Michigan. November 14, 1995. Also presented to the Department of Internal Medicine. Veteran Administration Medical Center. Allen Park, Michigan. March 19, 1996.

Legal Issues of Concern to Pharmaceutical Representatives. Detroit Pharmaceutical Representative Association. Southfield, Michigan. December 1, 1995.

The Future of American Health Care: A crystal Ball. Presented at Wayne State University School of Medicine for Primary Care Week. Gordon Scott Hall. Detroit, Michigan. September 27, 1995. Also presented to the following groups: Department of Ophthalmology Grand Rounds. Henry Ford Hospital. Detroit, Michigan. February 16, 1996. Glaxo-Wellcome Michigan Representatives Meeting. Ann Arbor, Michigan. March 27, 1996. Wayne State University School of Medicine. Gordon Scott Hall. Detroit, Michigan. February 14, 1997.

Elderly Law: A primer for Health Care Professionals. Presented to the Department of Geriatrics, Henry Ford Hospital. Detroit, Michigan. October 5, 1995.

An Ethical Dilemma: Informed Consent. Harper Hospital. Detroit, Michigan. February 28, 1996.

Violence in the Workplace. Walsh College. Troy, Michigan. March 8, 1996.

Law and Medicine: What they didn't teach you in Medical School-Part II. Department of Internal Medicine. Business Series for Residents. St. John Hospital and Medical Center. Detroit, Michigan. May 23, 1997.

Expert Testimony in Environmental Litigation. Presented for the Institute of Continuing Legal Education at the Michigan Environmental Planning and Litigation Update. MSU Management Education Center. Troy, Michigan. July 16, 1998.

Florida Laws and Rules
Michigan Osteopathic Association 101st Annual Postgraduate Convention & Scientific Seminar. Hyatt Regency Dearborn, Michigan. Saturday, May 20, 2000.

Lungs and the Listings
National Organization of Social Security Claimants' Representatives. Social Security Disability Law Conference Annual Spring Conference. Hilton at Walt Disney World Village. Lake Buena Vista, Florida. May 4, 2000.

Worker's Compensation for Occupational Medicine Physicians
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Gordon Scott Hall
Detroit Michigan. May 25, 2000.

Lungs and the Listings
State Bar of Michigan
Social Security Section Seminar
Park Place Hotel
Traverse City, Michigan. July 28, 2000.

Interesting War Stories from the Worlds of Medicine, Law and Actuarial Analysis
Michigan Actuarial Society
Hilton Inn
Southfield, Michigan. September 11, 2000.

Statistics in Environmental Litigation.
Environmental Litigation Committee
Environmental Law Section
State Bar of Michigan
Michigan National Tower. Lansing, Michigan February 17, 2001.

Worker's Compensation for Occupational Medicine Physicians
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 22, 2001.

Florida Laws and Rules
Michigan Osteopathic Association 102nd Annual Postgraduate Convention & Scientific Seminar. Hyatt Regency Dearborn, Michigan. Saturday, May 12, 2001.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 16, 2001.

Genetic Evidence in Paternity Cases: What the Lawyer Must Know
Presented by the Domestic Relations Committee of the Macomb County Bar Association
5th Floor Jury Room
Macomb County Court Building
Mount Clemens, Michigan June 4, 2001

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 15, 2001.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 18, 2002.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 23, 2002.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 5, 2002.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. November 11, 2002.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 3, 2003.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 4, 2003.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 4, 2003.

Worker's Compensation for Occupational Medicine Physicians and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 9, 2004.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 10, 2004.

Legal Issues for the Intern. Intern Orientation. Henry Ford Bi-county Hospital. June 16, 2004. Warren, Michigan.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 16, 2004.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 14, 2005.

Winning the Sick Building Case.
Michigan Trial Lawyers Association.
Shanty Creek Resort. Bellaire, Michigan. February 11, 2005.

The Second Most Important Lecture of Your Career. Michigan Chapter of the American College of Cardiology. William Beaumont Hospital Heart Center. Royal Oak, Michigan April 20, 2005.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 9, 2005.

Medical and Legal Considerations for the Occupational Medicine Physician.
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. July 15, 2005.

What the Occupational Medicine Physician Should Know about Toxic Tort Law.
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. July 22, 2005.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. November 7, 2005.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 6, 2006.

Radium City Town Hall Meeting
Panel participation
Occupational and Environmental Health (CM) 7860
Applebaum Hall
Wayne State University School of Pharmacy and Allied Health
Detroit, Michigan. February 1, 2006

Worker's Compensation Law and the Americans with Disabilities Act
Occupational and Environmental Health (CM) 7860
Applebaum Hall
Wayne State University School of Pharmacy and Allied Health
Detroit, Michigan. February 22, 2006

Proving and Disproving Causation in Environmental Litigation
Panel Participation
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan
Presented at the office of Ernest Chiodo P.C. Clinton Township, Michigan. February 23, 2006.

Interesting Answers to Complex Disability Problems.
Michigan Safety Conference.
Lansing Center, Lansing, Michigan April 18, 2006.

Legal Consideration in Medical Specialty Selection
Wayne State University School of Medicine –American Medical Association Student Chapter.
Wayne State University School of Medicine. Gordon Scott Hall. Detroit, Michigan. April 21, 2006.

Mold: State of the Law and Science
Joint Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan and the Michigan Industrial Hygiene Society.
Presented at the office of Ernest Chiodo P.C. Clinton Township, Michigan. May 1, 2006.

Case Studies in Forensic Medicine
Consulting Physicians P.C. Spring 2006 Medical Education Seminar
Holiday Inn South Convention Center. Lansing, Michigan May 3, 2006.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 8, 2006.

Toxic Exposure
GM National Benefits Center
Southfield, Michigan. June 12, 2006

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 14, 2006.

Carbon Monoxide: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at the office of Ernest Chiodo P.C. Clinton Township, Michigan. September 20, 2006.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. November 6, 2006.

Metal Working Fluids: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at the office of Ernest Chiodo P.C. Clinton Township, Michigan. November 14, 2006.

Asbestos: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice committee of the Environmental Law Section of the State bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on January 23, 2007. Presented at Varnum Riddering Schmidt Howlett LLP in Grand Rapids, Michigan on January 24, 2007.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 5, 2007.

Lead: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice committee of the Environmental Law Section of the State bar of Michigan.
Presented at the University of Detroit/Mercy Law School in Detroit, Michigan on March 12, 2007. Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on March 14, 2007. Presented at Varnum Riddering Schmidt Howlett LLP in Grand Rapids, Michigan on March 15, 2007. Presented at Wayne State University Law School in Detroit, Michigan on March 21, 2007.

Forensic Case Studies
Michigan Safety Conference
Devos Place. Grand Rapids, Michigan. April 17, 2007

Case Studies in Forensic Medicine
Consulting Physicians P.C. Spring 2006 Medical Education Seminar
Holiday Inn South Convention Center. Lansing, Michigan April 18, 2006.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 7, 2007.

Chromium: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on July 23, 2007.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 9, 2007.

Legal Considerations for Physicians when Patients Sign Out of Hospital Against Medical Advice.
Presentation to the medical staff of Henry Ford Bi-County Hospital.
Henry Ford Bi-County Hospital. Warren, Michigan. August 14, 2007.

Law, Medicine, and Industrial Hygiene
Presentation at the Annual Scientific Meeting of the Michigan Occupational and Environmental Medicine Association
Michigan State University Kellogg Conference Center
East Lansing, Michigan. September 28, 2007

Industrial Hygienist as a Consultant
Moderator of the Mini-conference
Michigan Industrial Hygiene Society
Michigan State University Management Education Center
Troy, Michigan. October 9, 2007

Respiratory Physiology
Department of Biomedical Engineering
Wayne State University
Detroit, Michigan November 7, 2007

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. November 12, 2007

Vapor Intrusions: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on November 20, 2007.
Presented at Varnum Riddering Schmidt Howlett LLP in Grand Rapids, Michigan on November 16, 2007.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 11, 2008

Vapor Intrusion: Law Medicine and Science. Present to the Illinois State Bar Association Environmental Law Section. Chicago, Illinois. February 29, 2008

Basics of Toxic Tort Law
Ave Maria Law School
Ann Arbor, Michigan March 12, 2008

Toxic Tort Law
Chicago Bar Association
Continuing Legal Education Series
Chicago, Illinois March 19, 2008 March 19, 2008

Forensic Medicine, Engineering, and Industrial Hygiene
Industrial Hygiene Section
Michigan Safety Conference
Lansing Convention Center
Lansing, Michigan April 15, 2008

Complex Disability Determinations
Michigan Occupational Health Nurses Section
Michigan Safety Conference
Lansing Convention Center
Lansing, Michigan April 15, 2008

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 19, 2008

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 11, 2008

Toxic Tort: What's Living in Your Sand Castle
Accident Fund 6th Annual TPA Conference
M.S.U. Henry Center
Lansing, Michigan. September 25, 2008

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. November 3, 2008

Respiratory Physiology
Department of Biomedical Engineering
Wayne State University
Detroit, Michigan November 10, 2008

Vapor Off-gassing: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on February 18, 2009.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 4, 2009

Employment Contracts for Cardiologists. Presented to the interventional cardiology fellows at St. John Hospital and Medical Center. Detroit, Michigan. June 1, 2009.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 3, 2009

Mold: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on March 30, 2010.

Forensic Toxicology
Presentation to the Midwest Association for Toxicology and Therapeutic Drug Monitoring 2010 Annual Meeting
Crowne Plaza Hotel, Milwaukee Wisconsin. April 30, 2010

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 10, 2010

Interesting Forensic Cases
Greater Peoria Claims Association
Edwards, Illinois. May 13, 2010

Vapor Intrusions: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at and the Chicago office of Barnes & Thornburg LLP and telecasted to their various offices on June 11, 2010.

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 25, 2010

Bioterrorism
Wayne State University
Department of Occupational and Environmental Health
Detroit, Michigan September 29, 2010

Toxicogenomics in Toxic Tort litigation
Environmental Law Section
State Bar of Michigan
Annual Meeting
Devos Place
Grand Rapids, Michigan September 30, 2010

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 7, 2011

Oil Spills: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the
Environmental Law Section of the State Bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on February 1, 2011.

Legal Aspects of Industrial Hygiene
American Industrial Hygiene Association – Chicago Section
Holiday Inn- Willow Brook, Illinois
March 16, 2011

Interesting Occupational and Environmental Forensic Cases
Loyola University Medical School
Department of Epidemiology and Preventive Medicine
Chicago, Illinois
March 18, 2011

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 9, 2011

Occupational Medicine Case Studies
Wayne State University
Department of Occupational and Environmental Health
Detroit, Michigan June 27, 2010

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. August 8, 2011

Panel Discussion: Environmental Law in Michigan after Trentadue.
Environmental Law Section
State Bar of Michigan Annual Meeting
Dearborn, Michigan. September 15, 2011

Low Speed Automobile Collisions.
Metro Detroit Office of Ernest Chiodo P.C.
Clinton Township, Michigan. November 3, 2011

Low Speed Automobile Collisions.
Royal Society of Medicine
London, UK. November 28, 2011

Low Speed Automobile Collisions.
City Place
West Palm Beach, Florida. January 14, 2012

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. February 27, 2012

Lead: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on March 6, 2012.

Interesting Forensic Cases
Saginaw Valley Adjuster Association
Frankenmuth, Michigan March 13, 2012

Low Speed Automobile Collisions
Arizona Insurance Claim Association
Phoenix, Arizona March 15, 2012

Worker's Compensation for Occupational Medicine Physicians
and Law and Medicine for the Occupational Medicine Physician
Department of Family Practice
Division of Occupational Medicine
Wayne State University School of Medicine
Detroit Michigan. May 7, 2012

Interesting Forensic Cases
Midwest Claim Conference
Grand Geneva Resort and Spa
Lake Geneva, Wisconsin May 17, 2012

Mold: Law, Medicine and Science
Presentation of the Environmental Litigation and Administrative Practice Committee of the Environmental Law Section of the State Bar of Michigan.
Presented at Ernest Chiodo P.C. in Clinton Township, Michigan on May 21, 2012.

Determining Injury Causation in Motor Vehicle Collisions
Auto-Owner's Nurse Case Managers and Adjuster
Auto-Owner's Headquarters
Lansing, Michigan. June 25, 2012

Interesting Forensic Cases
Grand Rapids Area Adjusters Association
Grand Rapids, Michigan
October 1, 2012

Interesting Forensic Cases.
The National Society of Professional Insurance Investigators-Illinois Chapter.
Downers grove, Illinois.
December 6, 2012

Introduction to Toxic Tort Law
Wayne State University Law School
Detroit, Michigan
February 11, 2013

Introduction to Toxic Tort Law
Environmental Law Club of Northern Illinois University Law School
DeKalb, Illinois
April 3, 2013

Forensic Industrial Hygiene
American Industrial Hygiene Association Indiana Section
Indianapolis, Indiana
April 10, 2013

Evaluating Injury Causation in Low Speed Automobile Accidents
Michigan Adjusters Association Seminar 113
Zender's of Frankenmuth
Frankenmuth, Michigan
May 3, 2013

Evaluating Injury Causation in Low Speed Automobile Accidents
AAA of Michigan
Southfield, Michigan
May 22, 2013

Biotechnology
The Strategy Symposium
University of Chicago Booth School of Business
Chicago, Illinois
April 25th and 26th, 2014

PUBLICATIONS:

Chiodo EP. Huff SH. Hadden DW. Increased Health, Life, and Disability Insurance Premium Costs: A New Class of Economic Damages in Toxic Tort Cases in Michigan. 17 Mich Env LJ, No 3. pp 3-6 (1999).

Hadden DW. Chiodo EP. Huff SH. The State of Trespass-Nuisance Law in Michigan. 18 Mich Env LJ. No 1. pp 8-11 (2000).

Chiodo EP. Genetic Evidence in Paternity Cases: What the Lawyer Must Know. Michigan Family Law Journal. Volume 30 Number 2. pp 11-12 (2001).

Chiodo Ernest P., Musial Joseph L, Robinson J Sia. An Error In Statistical Logic In The Application Of Genetic Paternity Testing. Journal OF Modern Applied Statistical Methods. Winter 2002. Volume 1, Number 1. pp 126-129.

Chiodo Ernest P., Huff Steven H. Attributable Risk Percent: A Unified Epidemiological Measure of Damages in Toxic Tort Cases. 21 Mich Env LJ. No 2. pp 12-14 (2004).

Chiodo Ernest P, Hadden Donnelly W, Huff Steven H. The State of Medical Monitoring Damages in Michigan after Henry v. Dow Chemical Co.26 Mich Env L J No 1, pp1-7 (2008).

Chiodo Ernest P. Economic and non-economic issues must be considered in toxic damage cases. Michigan Lawyers Weekly. June 1, 2009.

Toxicogenomics and Toxic Tort Litigation. 28 Mich Env L J No1 Pages 10-12. (2010).

Evaluating Injury Causation in Low Speed Automobile Collisions. November 7, 2011. Claims Journal.

Consider Medical Specialist Expertise Carefully in Crash Injury Cases. October 5, 2012. Claims Journal.

BOOKS:

Ernest P. Chiodo, M.D., J.D., M.P.H. Toxic Tort: A Guide to Toxic Substances Litigation in Michigan. Xlibris. Copyright 2002. Library of Congress Number 2002095046. ISBN Hard cover 1-4010-7725-0 Soft cover 1-4010-7386-7.

Ernest P. Chiodo, M.D., J.D., M.P.H. Toxic Tort: Medical and Legal Elements. Xlibris. Copyright 2004. Library of Congress Number 2003092354. ISBN Hard cover 1-4134-0537-1 Soft cover 1-4134-0536-3.

Ernest P. Chiodo, M.D., J.D., M.P.H., C.I.H. Toxic Tort: Medical and Legal Elements. Second Edition. Xlibris. Copyright 2007. ISBN Hard cover 1-4257-4962-3. Soft cover 1-4257-4961-5.

Ernest P. Chiodo, M.D., J.D., M.P.H., M.S., M.B.A., C.I.H. Bioterrorism. Xlibris. Copyright 2013. ISBN Hard cover 978-1-4797-8431-8. Soft cover 978-1-4797-8430-1. Ebook 978-1-4797-8432-5.

Ernest P. Chiodo, M.D., J.D., M.P.H., C.I.H. Toxic Tort: Medical and Legal Elements. Third Edition. Xlibris. Copyright 2013. ISBN Hard cover 978-1-4797-8434-9. Soft cover 978-1-4797-8433-2.

BOOK CHAPTERS:

Thomas W. Armstrong, Ernest P. Chiodo, Robert F. Herrick, and Christopher P. Rennix. Occupation Epidemiology. Chapter 6. The Occupational Environment: Its Evaluation, Control, and Management. 3rd edition. Copyright 2011 by the American Industrial Hygiene Association. ISBN 978-1-935082-15-6.

Crisis Management & Emergency Planning: Preparing for Today's Challenges
Chapter 13: Legal Considerations in Threat Response Management
Copyright 2014 by Taylor & Francis Group, LLC. ISBN 13:978-1-4665-5505-1.

PROFESSIONAL HONORS:

Co-author of the first place winning presentation at the 54th annual Providence Hospital Clinic Day. This was the first time that the Department of medicine had won the first place award in 19 years.

1991 Oakland Health Education Program Award for Research

FOREIGN LANGUAGE PROFICIENCY:

German - fluent
Mandarin Chinese - moderate conversational proficiency
Italian - minor to moderate conversational proficiency
Arabic - minor conversational proficiency

Ernest P. Chiodo, M.D., J.D., M.P.H., M.S., M.B.A., C.I.H.

TESTIMONY

Jovia Y. Chin vs. Madelyn M. Basnett
In the Circuit Court of Cook County, Illinois
County Law Department, Law Division
Case No. 09 L 007570
Deposition
May 9, 2011

Melissa McClellan vs. Laura Masbruch
In the Circuit Court of the 17th Judicial Circuit
County of Winnebago
Case No. 2010 L 252
Deposition
May 27, 2011

Valentin Castillo, Jr. vs. Dariusz Jan Las and Krzysztof
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 002025
Deposition
June 16, 2011

Deborah McClintic v. Barbara Templeman
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 012432
Deposition
July 22, 2011

Brad J. Carson xxx-xx-9340 vs. Metro Industrial Contracting, Inc and Zurich-American Insurance Co. of Illinois.
State of Michigan Department of Labor and Economic Growth
Workers' Compensation Agency
Deposition
July 25, 2011

Tammy Dang Do v. William F. Wangler
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 009125
Evidence Deposition
August 5, 2011

Jovia Y. Chin and Chee Chin Chong vs. Madelyn M. Basnett
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 007570
Evidence Deposition
August 12, 2011

Michael G. Moritz v. Oscar Carrera, et al.
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 08 L 8803
Deposition
August 19, 2011

Sam Aiello and Roxanne Aiello, Individually, and as next Friend and mother of Diane Lynn Aiello v. Speedwing Investment Company, a Michigan c0-partnership consisting of Otto Binggerer, James Williams, Jerry Borsard and Vernon Buchanan, as co-partners and d/b/a Pine Lake Manor Apartments
State of Michigan
In the Circuit Court for the County of Oakland
Case No. 08-091658-NO
Deposition
August 22, 2011

Jason Matteucci vs. EQ the Environmental Quality Company, a Michigan Corporation and EQ the Environmental Quality Company, a Michigan Corporation vs. Waste Management of Michigan, Inc. a Michigan corporation
State of Michigan
In the Circuit Court for the County of Wayne
Case No. 08-122650-NO
Evidence Deposition
August 30, 2011

Hyacinth Burgess v. Girish Patel
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 007554
Deposition
September 1, 2011

Christine Mosier-Peterson vs. Ronald Weiss & Great Lakes Working Dog Association
State of Illinois
In the Circuit Court of the Eighteenth Judicial Circuit Dupage County, Illinois
No. 09 L 1218
Deposition
September 23, 2011

Moore Living Trust d/b/a North Meadow Village Mobile Home Park and Hilltop Mobile Home Sales, v. Bobbie Vocke
State of Illinois
In the Circuit Court of the Eleventh Judicial Circuit Mclean County, Illinois
Case No. 08-LM-540
Deposition
September 23, 2011

Dana Henry vs. Edgar Gonzales and Alberto Gonzalez
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 10 L 3132
Deposition
September 30, 2011

Dennis McNamara SSN XXX-XX-2271 vs. Shank Coupland Long
State of Michigan Department of Labor and Economic Growth
Workers' Compensation Agency
Deposition
November 4, 2011

Hisar Moore vs. Cook County Housing Authority
State of Illinois
Illinois Workers' Compensation Commission
Case # 08 WC 50928
Deposition
November 9, 2011

Christine Mosier-Peterson vs. Ronald Weiss
State of Illinois
In the Circuit Court of the Eighteenth Judicial Circuit Dupage County, Illinois
No. 09 L 1218
Evidence Deposition
December 5, 2011

Lisa Labrake, Connie Nighswander v. CEI Michigan, LLC, a Michigan limited liability company
State of Michigan
In the Circuit Court for the County of Saginaw
Case No. 10-008493-NO-3
Deposition
December 13, 2011

Jesus "Jesse" Morales vs. Flavio Arana
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 10 L 000281
Deposition
December 20, 2011

Elizabeth A. Stefka vs. Laura Walker
State of Illinois
In the Circuit Court of the Twenty-Second Judicial Circuit
McHenry County, Illinois
No. 09 LA 187
Deposition
January 6, 2011

Patricia Burleson, et al. vs. Administrator, BWC, et al.
State of Ohio
Court of Common Pleas
Hamilton County, Ohio
BWC Claim No. 08-884623
Evidence Deposition
January 10, 2012

John Samp SSN XXX-XX-7616 vs. Decorative Panels, International, et al.
State of Michigan Department of Labor and Economic Growth
Workers' Compensation Agency
Deposition
January 24, 2012

Dennis R. Kocot SSN XXX-XX-6593 vs. Glawe, Inc. and its Insurance Carrier, Zurich-American Insurance Co.
State of Michigan Department of Labor and Economic Growth
Workers' Compensation Agency
Deposition
January 30, 2012

Justin Newcom vs. Ignacio Lopez
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 2010 L 009744
Deposition
February 24, 2012

People of the State of Michigan vs. Bruce Farley
In the Circuit Court for the County of Macomb
Case No. 11-2012 FH
Trial Testimony
March 20, 2012

Bryon Hardy; Husband of Diane Hardy (Deceased) v. Bigfoot Food Stores
Before the Workers' Compensation Commission in the State of Illinois
No. 04 WC 61267 with 11 WC 1642 & 11 WC 30001
Evidence Deposition
June 29, 2012

Beth Boidanis vs. Natalia McDonald
In the Circuit Court of Cook County, Illinois
County Department, Law division
No. 09 L 15148
Deposition
July 13, 2012

Maria Davila vs. Chicago transit Authority
In the Circuit Court of Cook County, Illinois
First Municipal District
No. 10 M1 300624
Deposition
July 26, 2012

Valentin Castillo, Jr. vs. Dariusz Jan Las and Krzysztof
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 002025
Trial Testimony
August 15, 2012

Jamies Konietzko, deceased, by Heather Hager vs. Center Guard Plumbing, Inc.
State of Illinois
Illinois Workers' Compensation Commission
Case # 10 WC 11213
Deposition
August 16, 2012

Nick Martinelli, Individually and as Special Administrator of the Estate of Laura Lee Martinelli, Deceased vs. Sherman Hospital, a corporation, Sherman Health System, a corporation, Stephen Grossman, M.D., Northwest Suburban Imaging Associates, S.C., a corporation, James L. Pinto, M.D. and James Leonard Pinto, M.D. & Associates, P.C., a corporation.
State of Illinois
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 09 L 9868
Deposition
August 23, 2012

State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Joseph Pedzinski vs. Erin EME
Case No. 10 L 8038
Deposition
September 14, 2012

State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Joseph Pedzinski vs. Erin EME
Case No. 10 L 8038
Evidence Deposition
September 27, 2012

State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
George Altonaga vs. Nizar Charania and Superior Taxi, Inc.
Case No. 11 L 928
Deposition
October 4, 2012

Steve Dunteman v. Caterpillar Inc.
State of Illinois
Illinois Workers' Compensation Commission
Case # 11 WC 40320
Evidence Deposition
October 25, 2012

Charlene Egan as Personal Representative for the Estate of Aaron M. Malone, III, deceased vs. John T. Golden, M.D.
State of Michigan
In the Circuit Court for the County of Macomb
Case No. 11-1013 NH
Deposition
November 6, 2012

Kristen Dunford v. Chicago Transit Authority
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 2010-L-005242
Deposition
December 7, 2012

Thomas L. Bice and Lucia A. Bice vs. Gemcraft Homes, Inc.
In the Circuit Court of Jefferson County, West Virginia
Case No. 10-C-72
Deposition
December 12, 2012

Armando Felix vs. Allied Insurance Company
In the Arbitration Matter of Felix Armando
Cook County, Illinois
Claim No. : 12B27580
Deposition
December 14, 2012

Juan Nelson v. Ahmed Tahir and Blue Ribbon Cab Company
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 2011-L-006811
Deposition
January 9, 2013

Ljiljana Pelivanovic vs. Our Lady of Resurrection
State of Illinois
Illinois Workers' Compensation Commission
Case # 10 WC 38521, 10 WC 38522, 10 WC 44279 & 12 WC 10959
Deposition
January 30, 2013

Jacobo Zarinana vs. Gabriel Alvarez
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 11-L-004947
Deposition
February 6, 2013

Dale and Louise Miller v Nagel Meat Processing
State of Michigan
In the Circuit Court for the County of Calhoun
Case No. 12-1067-NO
Deposition
February 18, 2013

Kristen Dunford v. Chicago Transit Authority
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 2010-L-005242
Evidence Deposition
February 27, 2013

Alexander Westbrook v. Kristin Ciesemier
State of Illinois
In the Circuit Court of DuPage County
Case No. 2010-L-001284
Evidence Deposition
March 1, 2013

Deborah Vyncke and Robert Vyncke v. Renato Ferreira
In the Circuit Court of the Twelfth Judicial Circuit
In and for Manatee County, Florida Civil Division
Case No: 2010-CA-00944
Division B
Deposition March 18, 2013

Michael Ault v. Longmont Business Partners, LLP d/b/a Best Western Firestone Inn and Suites and Third Party Defendant Ecolab, Inc.
In the District Court, Weld County, Colorado
Case No. 12cv283
Division 5
Deposition April 16, 2013

Illinois Workers' Compensation Commission
Pam Crusoe v. Edward Hospital
Case # 08 WC 52700
Evidence Deposition
May 2, 2013

Juan Nelson v. Ahmed Tahir and Blue Ribbon Cab Company
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 2011-L-006811
Deposition
May 9, 2013

Donna S. Werner and Marc Werner vs. Great Northern Insurance Company Inc., Chubb & Son, a division of Federal Insurance Company, Belfor USA Group, d/b/a Belfor Property Restoration, Aggreko, LLC, Dryco LLC, and Patten, Inc. as Defendants. Great Northern Insurance Company Inc., Chubb & Son, a division of Federal Insurance Company, Belfor USA Group, d/b/a Belfor Property Restoration, Aggreko, LLC, Dryco LLC, as Third-Party Plaintiffs, vs. Chagall, Inc., and Fabrick Power Systems, as Third-Party Plaintiffs.
In the Circuit Court of the Nineteenth Judicial Circuit, Lake County, Illinois.
Case No. 12 L 433
Deposition
May 10, 2013

Donna S. Werner and Marc Werner vs. Great Northern Insurance Company Inc., Chubb & Son, a division of Federal Insurance Company, Belfor USA Group, d/b/a Belfor Property Restoration, Aggreko, LLC, Dryco LLC, and Patten, Inc. as Defendants. Great Northern Insurance Company Inc., Chubb & Son, a division of Federal Insurance Company, Belfor USA Group, d/b/a Belfor Property Restoration, Aggreko, LLC, Dryco LLC, as Third-Party Plaintiffs, vs. Chagall, Inc., and Fabrick Power Systems, as Third-Party Plaintiffs.
In the Circuit Court of the Nineteenth Judicial Circuit, Lake County, Illinois.
Case No. 12 L 433
Deposition
May 24, 2013

Lisa Parr, Individually and as Next Friend to Her Minor Daughter, E.D., and Robert "Bob" Parr vs. Aruba Petroleum, Inc., Burlington Resources Oil &Gas Company LP (A subsidiary of ConocoPhillips), Encana Oil & Gas (USA) Inc., Halliburton Company.
In the County Court At Law No. 5, Dallas County, Texas.
Cause No. CC-11-01650-E
Evidence Deposition
June 25, 2013

Lori Budrow, as Wife of Kenneth T. Budrow, JR., Deceased vs. Oak Spur Hills Apartments Associates, L.L.C. d/b/a Cherry Creek Apartments, et al.
In the Circuit Court of St. Louis County
State of Missouri
Cause No. 09SL-CC00990
Division 8
Evidence Deposition
June 28, 2013

Andrew Hopper vs. Southern Air, Inc. and Insurance Co. of the State of Pennsylvania.
State of Michigan
Department of Labor & Economic Growth
Workers Compensation Agency
Evidence Deposition
July 9, 2013

Athena Christus v. Abrida Kassim and Ghulam M. Kassim
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 09 L 006126
Deposition
July 15, 2013

David Prim, as Administrator of the Estate of Susan Prim, and Brodie Prim and Tyler Prim, Individually v. Capital Improvement Board, City of Indianapolis, Event Services, Inc., Le Maitre Special Effects (USA), Inc., d/b/a Ultratec Special Effects, Inc., Next F/x, Inc., Pacers Basketball, LLC d/a/a Conseco Fieldhouse, Pyro Spectaculars, Inc., Res Specialty Pyrotechnics, Inc., World Wrestling Entertainment, Inc., Zenith Pyrotechnology, Fireworks West International, Marion County Convention and Recreational Facilities Authority, Stage F/x, Inc. and Sichuan Onward International Trade Co., LTD.
State of Indiana
County of Marion
Marion Superior court No. 5
Cause No. 49D05-0904-CT-016339
Deposition
July 18, 2013

Jimmy R. Rice v. Sloan Valve Company
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 10 L 4071
Deposition
July 19, 2013

Justin G. Newcombe vs. L.E. Jones Company
State of Michigan
Department of Labor & Economic Growth
Workers Compensation Agency
Evidence Deposition
August 6, 2013

Physicians Healthsource, Inc., an Ohio corporation, individually and as the representative of a class of similarly-situated persons v. Stryker Sales Corporation, Stryker Biotech L.L.C., Stryker Corporation, Howmedica Osteonics Corp. and John Does 1-10.
In the United States District Court for the Western District of Michigan
Civil Action No.: 1:12-cv-00729-RJJ
Deposition
August 15, 2013

Tammy Maida vs. Mary Gill
State of Illinois
In the Circuit Court of the Eighteenth Judicial District
Dupage County, Illinois
Case No. 11 L 000010
Deposition
August 23, 2013

Joseph Villarreal v. German Valle-Guerrero,
Jason Williams and Barbara Campbell
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 07 L 004129
Deposition
September 6, 2013

Athena Christus v. Abrida Kassim and Ghulam M. Kassim
State of Illinois
In the Circuit Court of Cook County
County Department, Law Division
Case No. 09 L 006126
Evidence Deposition
September 6, 2013

James Willis, individually, and on behalf of Jacqueline Willis, deceased, and on behalf of Julianne Willis, a minor child vs. BP Corporation North America, Inc., BP Products North America, Inc., James Blaise, M.D. Boatwright, J. Lewis Sutton, Floyd Thomas, Ronald W. Ginson, Clifford H. Carver, and John Does 1-20
In The Circuit court of Jackson County, Missouri at Independence
Case No. 1216-CV02408
Deposition
October 11, 2013

William Estes, Jr. and Claudia Estes vs. Holiday CVS, LLC, d/b/a CVS Pharmacy #3229; and CVS EGL Atlantic Neptune Beach, FL., LLC, d/b/a CVS Pharmacy #3229; Douglas Allen Swartz, M.D.; Integrated Community Oncology Network, LLC., d/b/a McIver Urological Clinic; and First Coast Urology, P.A.
In the Circuit Court of the Fourth Judicial Circuit of Florida, in and for Duval County
Case No.: 16-2012-CA-007406
Deposition
December 6, 2013

Brian Kahn vs. The Dewey Group; Storage Etc., LLC; B Mortgage Acceptance Corp.; Sky Terrace Investors, LLC; SE Sky Terrance, LLC; SE Sky Spe, LLC; Storage Etc. Manager, LLC.; Angelo Capital Realty, LLC; Laurent Opman; Laurent Opman Living Trust; Bruce Rothman; Rothman-Kaye Family Trust; Barbara Andrea Kaye; Sky Dewey, LLC; John Dewey; Dewey Family Trust; Rebecca Dewey; Dewey-Koar, LLC; SE Lopez 1, LLC; Laurent A. Opman; and Greg Houge.
Superior Court of the State of California for the County of Los Angeles
Case No. BC454443
Deposition December 12, 2013

Illinois Workers' Compensation Commission
James Deans v. Medlogic
Case # 13 WC 014230
Evidence Deposition
January 22, 2014

Beth Boidanis vs. Natalia McDonald
In the Circuit Court of Cook County, Illinois
County Department, Law division
No. 09 L 15148
Evidence Deposition
January 24, 2014

Lindsay Byers v. Jose Morillo, and Chicago International Trucks, L.L.C
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 10 L 7894
Deposition
January 24, 2014

Cassie Terry et al. vs. Carnival Corporation, d/b/a Carnival Cruise Lines, in personam
United States District court
Southern District of Florida
Miami Division
Case No. 13-20571-CIV-Graham/Goodman
Deposition
January 30, 2014

Michael C. Silvius v. Bartlett Fire Protection District
Before the Illinois Workers' Compensation Commission
Case Numbers 13 WC 37661, 13 WC 37662, 13 WC 37663
Evidence Deposition
February 12, 2014

Dorothy Smith vs. Tracy Thomas Bartholomew, Ultimate Trucking, LLC, and Security National Insurance Company
Civil District Court for the Parish of Orleans, Division C
No. 2013-1177
Deposition
February 12, 2014

Derek Stephens vs. Eric Harland a/k/a Eric Harman
In the Circuit Court for the Tenth Judicial Circuit Peoria County, Illinois
Case No. 11-L-88
Evidence Deposition
February 13, 2014

Cassie Terry et al. vs. Carnival Corporation, d/b/a Carnival Cruise Lines, in personam
United States District court
Southern District of Florida
Miami Division
Case No. 13-20571-CIV-Graham/Goodman
Trial Testimony
February 28, 2014

Brian Kahn vs. The Dewey Group; Storage Etc., LLC; B Mortgage Acceptance Corp.; Sky Terrace Investors, LLC; SE Sky Terrance, LLC; SE Sky Spe, LLC; Storage Etc. Manager, LLC.; Angelo Capital Realty, LLC; Laurent Opman; Laurent Opman Living Trust; Bruce Rothman; Rothman-Kaye Family Trust; Barbara Andrea Kaye; Sky Dewey, LLC; John Dewey; Dewey Family Trust; Rebecca Dewey; Dewey-Koar, LLC; SE Lopez 1, LLC; Laurent A. Opman; and Greg Houge.
Superior Court of the State of California for the County of Los Angeles
Case No. BC454443
Rule 402 Evidence Hearing
March 20, 2014

James P. Bowman (Plaintiff) and Baldwin & Lyons and Protective Insurance Company (Intervenors) v. Carl W. Collins and Yvette & Robert Trucking, LLC
United States District Court
Southern District of Indiana
New Albany Division
Case No. 4:12-CV-0146-SEB-DML
Deposition
April 3, 2014

Brian Kahn vs. The Dewey Group; Storage Etc., LLC; B Mortgage Acceptance Corp.; Sky Terrace Investors, LLC; SE Sky Terrance, LLC; SE Sky Spe, LLC; Storage Etc. Manager, LLC.; Angelo Capital Realty, LLC; Laurent Opman; Laurent Opman Living Trust; Bruce Rothman; Rothman-Kaye Family Trust; Barbara Andrea Kaye; Sky Dewey, LLC; John Dewey; Dewey Family Trust; Rebecca Dewey; Dewey-Koar, LLC; SE Lopez 1, LLC; Laurent A. Opman; and Greg Houge.
Superior Court of the State of California for the County of Los Angeles
Case No. BC454443
Trial Testimony
April 11, 2014

Brenda White vs. Stanley Zwiren
In The Circuit Court of the Fifteenth Judicial Circuit in and for Palm Beach County, Florida
Case No. 50 2013 CA 011795 XXXX MB AD
Deposition
April 17, 2014

Scott Maghakian vs. National Union Fire Insurance Company of Pittsburg, PA.
United States District Court Northern District of Indiana Fort Wayne Division
Case No. 1:13CV024
Deposition
April 25, 2014

Tonya T. Sheldon, individually and as Administrator of the Estate of Shawn M. Sheldon vs. Bosserman Aviation Equipment, Inc. and Terry Bosserman.
In the Common Pleas Court of Hancock County, Ohio
Case No.: 2011-CV-288
Deposition
May 1, 2014

Cheryl Pugh vs. Evelyn Kuryla
In the Circuit Court of the Twelfth Judicial Circuit
Will County, Illinois
County Department, Law Division
No. 12 L 0002
Deposition
May 28, 2014

Severino Enriquez and Barbara Enrique v. Guillermo J. Malave Castro and Associated Electrical Services, Inc.
In the Circuit Court of the 17th Judicial Circuit in and for Broward County, Florida
Civil Division
Case Number 10-047812 CA 21
Deposition
June 5, 2014

Renuka Patel v. Tim Hoffman
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 12 L 6721
Deposition
July 10, 2014

Corey Terrell Green vs. Outstanding Transport, Inc. and John Jackson
Supreme Court of the State of New York
County of Kings
Index No.: 12463/10
Trial testimony
July 11, 2014

Nancy Cohn vs. Norman Meczyk and Sharon P. Krone
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 08L013732 Consolidated with 10L007288
Deposition
August 6, 2014

Carmen Acosta v. Shelly Thompson
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 20 L3398
Deposition
August 13, 2014

Illinois Workers' Compensation Commission
Patricia Nielson v. St Peter & Paul Church
Case # 03 WC 29556 & 07 WC 42098
Evidence Deposition
September 26, 2014

Kelvin Brandon vs. Steven Alfred, individually and as agent of Chicago Transit Authority, a municipal corporation, Chicago Transit Authority, a municipal corporation, individually, and Jose Garcia, individually
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 08L013732 Consolidated with 10L007288
Deposition
October 1, 2014

Jodelle L. Kirk vs. Schaeffler Group USA, Inc., et al.
In The United States District Court for the Western District of Missouri
Southwestern Division
No. 3:13-CV-05032-DGK
Deposition
October 6, 2014

Patricia Jolly v. Dominick Martino Bolden, Monique Lashun Caldwell, C&M Transportation & Limousine, LLC, and American Couuntry Insurance Company.
State of Michigan
In The Circuit Court for the County of Oakland
Case No. 2013-133123-NI
Evidence Deposition
October 7, 2014

Severino Enriquez and Barbara Enrique v. Guillermo J. Malave Castro and Associated Electrical Services, Inc.
In the Circuit Court of the 17th Judicial Circuit in and for Broward County, Florida
Civil Division
Case Number 10-047812 CA 21
Trial Testimony
October 10, 2014

Michael Rosenberg v. Victoria M. Stedt
In the Circuit Court of the 15th Judicial Circuit in and for Palm Beach County, Florida
Case No.: 502013 CA 005566MB AD
Evidence Deposition
October 24, 2014

He Ma v. Sigma Management Company, et. al.
Superior Court of New Jersey
Law Division-Gloucester County.
Docket No. Glo-L-339-13
Evidence Deposition
November 7, 2014

Ronald D. Getz v. Allstate Insurance Company
In The Circuit Court of the Ninth Judicial Circuit
In and for Orange County, Florida
Case No.: 2012-CA-7586
Discovery Deposition
November 14, 2014

Ronald D. Getz v. Allstate Insurance Company
In The Circuit Court of the Ninth Judicial Circuit
In and for Orange County, Florida
Case No.: 2012-CA-7586
Evidence Deposition
November 14, 2014

Kristy Hardville v. Spencer Looney, A. Butler & Associates, L.L.C. and American Country Insurance Company.
In the Circuit Court for the County of Wayne
State of Michigan
Case No. 13-012958-NI
Deposition
December 15, 2014

Carol Cross v. Tegora Cab Corporation, Choice Taxi Association, Habtu Baraki
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 13 L 003080
Deposition
December 17, 2014

Lisa Tindle v. Modine Manufacturing Company
United States District Court for the Western District of Missouri
Civil Action No. 2:14cv-04061-NKL
Deposition
January 22, 2015

Tracey Bloxham, Individually and as Personal Representative of the Estate of Adrian Larkin Merrill (aka "Jay Merrill"), Cherie Merrill, Joshua Merrill, Jamie Merrill, Cynthia Stumbo, and Wendy Perkins v. Pro-Point, Inc., Bridgepoint Systems, Karcher North America, Inc. Does 1-100.
Case No. A-12-671324-C
District Court
Clark County Nevada
Dept. No 1
Deposition
February 2, 2015

Kristina Riggs v. Luz Miriam Ramirez and Government Employees Insurance Company
In the Circuit Court of the 16th Judicial Circuit in and for Monroe County, Florida
Case No. 12 CA 217 P
Evidence deposition
February 3, 2015

Carmen Acosta v. Shelly Thompson
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 20 L3398
Evidence Deposition
February 5, 2015

Clifford A. Ekkert v. Village of Oak Brook Fire Department
Illinois Workers' Compensation Commission
Case # 13 WC 29508
Evidence Deposition
February 12, 2015

Lorinda Kronenberg v. Nick Phillips and Montgomery Cab, Inc. a/k/a A Cab of Aurora
In the Circuit Court of for the 18th Judicial Circuit DuPage County, Wheaton, Illinois
No. 2011 L 001291
Deposition
February 19, 2015

Ronald Wyatt v. Honeywell
Illinois Workers' Compensation Commission
Case # 08 WC 37513 and 09-WC-22941
Evidence Deposition
February 26, 2015

John Vasquez v. Yuriy Petrusyak and Halyna Hauk
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 12 L 11144
Deposition
March 12, 2015

Keith Bounds and Megan Lee v. Chicago Transit Authority and Keith Brown
In the Circuit Court of Cook County, Illinois
County Department, Law Division
No. 11 L 004399
Deposition
April 2, 2015

Clifford A. Ekkert v. Village of Oak Brook Fire Department
Illinois Workers' Compensation Commission
Case # 13 WC 29508
Evidence Deposition
April 16, 2015

Robert Fults SSN XXX-XX-9424 v. Marine City Police Department and Home Insurance Company c/o Michigan Property & Casualty Guaranty Association
State of Michigan Department of Licensing & Regulatory Affairs
Workers' Compensation Agency
Evidence Deposition
April 21, 2015

Anthony Perrone v. Thomas Perkins
United States District Court
Western District of New York
Case No. 11-cv-64111 (CJS)
Deposition
April 23, 2015

Scott Maghakian v. Cabot Oil & Gas Corporation, Factory Equipment Excavating
United States District Court
For the Middle District of Pennsylvania
Civil Action No. 3:12-cv-02346-UN4
Deposition
April 28, 2015

Cheryl Pugh vs. Evelyn Kuryla
In the Circuit Court of the Twelfth Judicial Circuit
Will County, Illinois
County Department, Law Division
No. 12 L 0002
Evidence Deposition
May 6, 2015